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The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

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Dallas, Texas

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THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY EIGHT

The Role of Organ Systems and Age in Periodontal Disease

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IF those who through the years have written and spoken on the etiology of periodontal disease are to be believed, one should think in terms of local and systemic factors. The usual connotation is that these are extreme opposites and that they may or may not be related to each other. Perhaps this is not so. It would appear that those who constantly demand a "cause" for periodontal disease expect it to be found in the so-called "systemic" area. And yet, as one searches even the modern literature, it is obvious that there is little appreciation of normal body system activity as related to both dental health and dental disease. Such information would be of vital importance in assessing systemic variations associated with periodontal disease.

It is my purpose to appraise such factors in the light of the clinical expression of periodontal disease and the organ system changes in health as it moves toward senescence. In so doing it is hoped that there will be indications as to what areas of research might offer promise in bringing about a better understanding of this disease.

There is no need for defining the term periodontal disease in this discussion, since all destructive processes affecting the periodontium, both local and general, must be assumed to come under the influence of changes of time.

The rather general inference that "systemic factors," as referred to in relation to periodontal disease, represent changes beyond physiologic tolerance and are, therefore, referable to clinical disease is open to serious challenge. This is so since the vast number of clinically healthy patients who demonstrate periodontitis give no reliable evidence of serious impairment of organ system activity. Yet changes in the milieu of the body are known to occur in health and to be related to very complex endocrine activity and that this interplay is simultaneously influenced by the changes of time. This, then, is the problem. Local "triggering influences" acting upon a complex and variable physiologic template are able to produce local destructive changes in the periodontium. It is to the variations in this "physiologic template" that we must direct our attention. In seeking answers to the problems at hand, great care must be taken in the formulation of the questions to be posed. In the design of experiments we cannot ask "what

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causes periodontal disease" since the source of such information, the human organism, can answer only questions to which it can say "yes" or "no." We must, therefore, inquire as to the role of individual structures, substances and circumstances, if we are gradually to understand this problem. We can, for example, design an experiment which poses the question—"does cortisone play any role in the inflammatory process that affects the periodontium in periodontal disease?" To this there is an answer and we shall hear more of this later.

BODY SYSTEM INFLUENCES

Functional Organ Activity. It is an accepted fact that body physiology taken in its general sense has an adaptive capacity and that this capacity decreases with the passage of time. Perhaps an analysis of some of these changes will shed light on the presently vague "systemic factors" in periodontal disease. Such an approach seems indicated since there is evidence that the incidence and severity of periodontal disease generally increases with the changes of time. Of great difficulty in such an analysis is the problem of defining the difference between senescence and disease. This can perhaps best be done by applying the "uniform subject selection criteria" of Falzone and Shock[1] to the changes that occur with the passage of time in a person for whom all diagnosable disease, which might affect the organ system in question, has been ruled out. Such an individual may then be assumed to present "the healthy normal subject." The criteria of Falzone and Shock[1] require three types of information—these are:

(1) The resting state of the internal environment

(2) The resting organ functions that maintain this state

(3) The reserve or maximal capacities of these organ systems and their ability to defend the internal environment against displacements induced by stress.

Regarding this internal environment, it is a basic principle in physiology that the function of most organ systems is to maintain the constancy of this state. This is the principle of homeostasis as advanced by Bernard,[2] Cannon,[3] and others.

We should now, for a moment, consider the anatomical site which we wish to explore in the face of physiologic changes that occur with time. The maxilla and mandible with their associated structures are in this area. It is not a critical zone in relation to total health; neither does it represent an important site in terms of total body economy. May we assume, therefore, that the subtle functional changes associated with decreased physiologic adaptability would affect these structures as a specific target, and if so, where, in the priority listing of such target areas, should the maxilla and mandible with their associated structures be placed? An answer to a part of these queries is apparent to all; certainly the cells and tissues of the oral areas live in the internal environment or milieu of the body, therefore, they must be affected by alterations which occur within this homeostatic environment. Some of these will be discussed at this time, particularly as they relate to the changes of age. As to the selectivity and specificity of the area, one can at present only speculate.

The functional interplay between organ systems is most complicated, and to demonstrate some of the physiologic alterations with age only one organ, but a most important one, will be considered—the kidney. You will recall that all arterial blood reaching the kidney passes through the glomeruli and that about 20 per cent is filtered through the membranes of these glomeruli. Of this 20 per cent, over 99 per cent is reabsorbed by the tubules, together with other substances, while waste products are excreted. The external product is urine of variable composition, and the internal component is blood plasma of constant composition. Because of the specificity of these values it is possible to assess kidney integrity. One indicative measurement can be obtained by diodrast clearance. Using this

test, Davis and Shock[4] have shown that renal plasma flow decreases from 600 cc. per minute in the 20-29 year age group to about 300 cc. per minute in the 80-89 year age group. Decrease in the glomerular filtration rate and other alternations can be demonstrated by means of other reliable tests.

At this point two important considerations are imperative. Falzone and Shock[1] have theorized that physiological functions and reserve capacities can be reduced enormously without change in the internal environment under resting conditions and that the older individual demonstrates a significant decrease in reserve capacity of organ systems, making him more vulnerable to the stresses of living, both emotional and physical, than the young.

In order that these points may have significance in deductions to follow, we must ask what, from a histological and histochemical point of view, happens when these changes occur? It would appear that functional loss of an organ system implies either a loss of active protoplasm or a qualitative difference in the protoplasm. Lowry and Hastings[5] have shown that the composition of protoplasm is relatively constant and does not change materially with age, whereas a variable weight loss of many organs, accompanied by widespread cell shrinkage, cell loss, and relative and absolute fibrosis, occurs with age. These data have led to speculation as to whether intercellular material or product passively replaces cells which shrink and die as they grow older, or the cells die because of a literal "strangulation" referable to aging of connective tissue which loses its permeability.

Regardless of the answers to these questions, it is clear that all body systems undergo retrograde changes with age. There is a decline in cardiac output, and pulmonary function changes markedly with time. Further, the "slowing down" of older individuals, exhibited in their speech, gait and cerebration, can be explained, at least in part, by the decreased oxidative metabolism as shown by a progressive lowering of the basal metabolic rate.

From studies of such changes Shock[6] has concluded that the aged individual is usually able to maintain uniformity of the internal environment. However, when increased demands are placed on a number of organ systems, impairment of function can often be detected. Thus, the older individual demonstrates a reduction in reserve capacity which makes him more vulnerable to the stressors of daily life than the young. Of importance, however, are the striking variations in the vulnerability of individuals in this regard, indicating that chronologic age does not necessarily parallel the changes of time.

These data are meaningful in the periodontal problem only if it can be shown that the areas in which we have an interest are affected. Before this question can be answered there is another aspect of the problem that must be considered.

HORMONAL INTERPLAY

It has been indicated previously that body physiology has an adaptive capacity and that this capacity decreases with the changes of time. We must also recognize the ability of the organism to adapt within limited ranges in response to immediate local stressors. It is with this mechanism that we must now be concerned. The fluctuation is largely under the influence of hormones, which are specific chemical substances produced by the endocrine glands and secreted into the blood, among other things, to regulate and control organs and organ systems.

Under the impact of stressors a stress reaction occurs which is represented by certain specific organ changes and the production and release of certain hormones. These organ changes are initially in the pituitary and adrenal glands although other organs are directly and indirectly affected. One must also remember the effect of these phenomena on processes of altered or accentuated activity. Normally a stressor applied locally tends to induce inflammation, but the nature and character of the resultant

process is seriously altered when the entire body is under stress. Selye[7] has defined stress as "the state manifested by a specific syndrome which consists of all the nonspecifically induced changes within a biologic system." Such a definition indicates the preciseness of the reaction and the nonspecificity of the cause.

We need not consider the details of the General Adaptation Syndrome and its integral aspects, but in this discussion the relationship between the General Adaptation Syndrome and the Local Adaptation Syndrome is of greater importance. Local destructive processes represent final stages of exhaustion of regional adaptation. Numerous such processes can operate simultaneously in various areas of the body, and in proportion to their intensity and extent may evoke the general adaptive response. Only when the whole organism is exhausted, as through accelerated aging caused by stress, does the terminal stage of exhaustion of the General Adaptation Syndrome manifest itself.

Now let us return to the local destruction process—in this context—periodontitis or inflammation of the periodontium. The release of adrenalin by the adrenal medulla as part of the general adaptive response is well known. Of equal importance is the release of an identical hormone—nor—adrenalin by nerves in peripheral areas. This permits selectivity of action in a localized area when an immediate adaptive response is needed. As a counterbalance these nerves also release acetylcholine which, in essence, controls the concentration and thus the action of the adrenalin. These regulators can directly induce adrenal-cortical activity to influence the local reaction and the inflammation. The exact mechanism for this response is, at present, under intensive study.

Of the variety of hormones produced by the adrenal cortex some tend to inhibit inflammation—others to enhance it. The anti-inflammatory corticoids, called A-C's, are chiefly cortisone and cortisol. The pro-inflammatory corticoids, called P-C's, are mainly aldosterone and desoxycorticosterone. It must be remembered that these hormones have other functions as well, for example, the anti-inflammatory corticoids also elevate blood sugar, hence are often called gluco-corticoids while the pro-inflammatory corticoids influence mineral metabolism and are sometimes referred to as mineral corticoids.

The way in which these hormones act generally or locally is dependent upon many conditioning factors, some internal and others external. One of the important external conditioning factors is diet, another, stress itself. For example, the same amount of cortisone which inhibits inflammation in a person who has just undergone a major physical trauma will have little or no effect on a healthy person.

NATURE OF THE LOCAL PROCESS

There is little question as to the nature of the process which motivates and sustains periodontitis. It is the exudative form of inflammation. I am convinced that this is the underlying mechanism in all aspects of the disease even though, as a clear-cut process, it is sometimes intermingled with other reactions such as in pregnancy gingivitis, or submerged in accentuated degenerative changes as in periodontosis.

The process of inflammation, although complicated, is fairly well understood so that this discussion will deal with the ways and means by which it is influenced directly by hormonal activity and indirectly by the physiologic changes of time.

Any stressor, such as bacteria or calculus, acts directly upon a target area, such as the gingival tissue, and indirectly upon the pituitary adrenal complex. The exact mechanism by which this latter effect is produced is not completely understood. This action evokes ACTH which increases the output of anti-inflammatory corticoids. This shifts the balance in favor of anti-inflammation. These hormones, cortisone and cortisol, prevent fibroblastic proliferation. Simultaneously the adrenal cortex, perhaps under influence of STH (growth hormone) raises the output of pro-inflam-

matory corticoids to re-establish hormone balance. The time involved in this transfer of values is dependent upon many factors—particularly the nature of the "triggering mechanism." It is important to know that the pro- and anti-inflammatory corticoids are initiated and sustained, as determined by blood concentration, through independent mechanisms. There are plentiful data available to explain the detailed chemistry of these reactions but this is not a necessary part of this discussion.

To associate these matters directly with periodontal disease is not a simple matter since it must be done in relation to limited data accumulated in the study of other somewhat similar diseases.

It is true that many diseases are the result of direct action of particular agents, such as bacteria, viruses, and poisons. Others, however, are not the result of a single cause but develop from the body's response to an unusual situation. What might be a usual circumstance can become unusual if environment and conditioning mechanisms, such as aging, are additional factors to overcome.

As a rule, inflammation is a very useful reaction; indeed it is a necessary response wherein the vascular and exudative aspects are reoriented to adjust to a local irritation in order to bring about repair. It is well known, however, that sometimes the response is excessive and quite out of proportion to a relatively innocuous irritant. Such a response is, in essence, disease itself. Since we have seen that inflammation is regulated by adaptive hormones, it could be postulated that a disturbance in the local adaptation syndrome may in reality be the prime cause of the disease. This becomes particularly meaningful when the peculiar environment of the periodontium is such that destruction of tissue and tissue relationships cannot be re-established in their original state. In this connection you will recall my remarks concerning the role of environment in converting a usual into an unusual situation.

The problem becomes most difficult when one remembers the area of the oral regions in relation to total body economy and its non-critical nature.

AREAS IN WHICH STUDY INDICATED

The literature is replete with references to the influence of system and organ diseases in periodontitis, particularly those affecting the endocrine glands. But these, for the most part, speak of gross aberrations such as are seen in pregnancy, diabetes and hyper-parathyroidism. It would appear that the role of endocrine glands and the hormones they produce in periodontal disease is more important in the local response and is quite unrelated to the clinical disease entities so often mentioned.

It seems obvious that the lesion in periodontitis and periodontosis is locally induced and sustained by any one or a combination of "triggering mechanisms" well known to all. The facility with which they can occur may be related to the efficiency of the local adaptive mechanisms which contribute an important regulating device in inflammation—the process which is induced in periodontitis. It can be reasoned that in limited instances the destructive lesion may not display the conventional clinical signs of inflammation, degeneration being the dominant reaction. This would most likely occur when a series of local adaptive responses are simultaneously in operation, thus depleting the General Adaptive Capacity. To say then that endocrine activity is important in periodontal disease is indeed accurate; however, this is within subtle ranges far short of the clinical expression of gross diseases.

Once the initial lesion is evoked and becomes clinically discernible, tissue is destroyed and tissue relationships are impaired and because of the environmental status of the periodontium cannot be completely restored.

The influence of the changes of time may be of greatest importance in eventually establishing a biologic template which is

quite susceptible to the local "triggering mechanism" and hence the initiation of the disease. It is reasonable to assume that these local "triggering mechanisms" are of prime importance in the problem. If they can be eliminated it would appear that the chain of events described would probably not occur. However, this is not possible. Further, circumstances existing or occurring in the young individual, innocuous at the time, may become more serious as the normal changes of time take place.

The influence of aging in the periodontal patient must be recognized in our thinking and in our studies. The question is "how important is this factor"? The complete answer to this will only come after the matter has been diligently appraised.

At the very beginning it must be remembered that the rapidly emerging view that gerontology is of great importance in clinical medicine will lead many research workers into the general area. We in the dental profession must not only be aware of their efforts and results but must complement them by planned investigations into problems more directly related to the oral regions.

Vogt and Vogt[8] have shown that the functional and morphologic alteration associated with aging is manifestly different in each of the hundreds of different cell types and that the aging process always leads to the death of the cell. This study could well be extended to the structures of the periodontium, particularly the cells of epithelial cuff. Tissue culture and ultraviolet resorption are possible tools for such an effort. Birren and Wall[9] have demonstrated that in the normal course of aging the metabolic activity of peripheral nerves is markedly disturbed, even to the extent of causing a decrease in fiber components. Again, this could be of great importance in the matter of muscular activity of the jaws and indirectly, at least, to the problems of traumatogenic occlusion.

One must, of course, mention the need for detailed study of the changes that occur in bone with age. Certainly there is abundant evidence that the mandible and maxilla are most vulnerable in this regard. Yet, these are gross observations. Histological, histochemical and tissue culture studies of these tissues in a controlled age study are certainly indicated.

Of perhaps greatest importance is the need for longitudinal studies, bringing together the clinician and laboratory worker to assess the clinical changes in the periodontium that occur with age in relation to biologic alterations in the structure of the periodontium.

The mature point of view toward human aging would seem to be a balance between optimism and realism and the avoidance of excess stress on limitations of aging. Certainly we cannot avoid the implications of the influence of the changes of age in periodontal disease. Indeed, I, for one, suggest we consider periodontal destruction a problem in gerontology, at least in part, until the effects of the changes of time have been shown to be innocuous in this regard. This, I am certain, will never come to pass.

CONCLUSIONS

It is suggested that all who have an interest in the periodontal problem critically examine the obvious association of periodontitis and aging. This seems a most reasonable approach in the light of the increasing emphasis on geriatric medicine.

There is an awakening of thought in the direction that some of the changes that occur with time may be modified and that basic underlying changes which lead to the diseases of the older person may be recognized and treated.

We must recognize that periodontitis and periodontosis occur, for the most part, in clinically healthy people who, considering the age frequency of the disease, offer little except a less rigid physiologic template as a likely source for the answer to this problem.

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Tooth Malpositions and Periodontal Pathosis: An Evaluation of Etiology and Considerations in Treatment

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PERIODONTAL disease often produces a loosening and change in position of teeth. Periodontal therapy includes adjunctive measures for the repositioning of migrated teeth. These interventions have been termed "minor tooth movements." Reports of individual cases and technique methods are numerous. There is a relative paucity of literature concerning the rationale for these movements.

Good practice requires a combination of knowing how and why; and an understanding of the broad purposes and basic implications of treatment. Such an understanding is engendered by clinical experience, historical perspective and knowledge of basic science. In this vein the historical background of minor tooth movement is introduced; the periodontist's area of responsibility is defined; occlusion, tooth wandering and minor movements are considered in terms of tissue response; criteria and indications for treatment, and anticipated benefits of therapy are enumerated. A subsequent paper will deal with specific measures for the repositioning of teeth.

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HISTORICAL BACKGROUND

Minor movements of adult teeth to improve periodontal health have been performed for centuries. Fauchard (1723) treated loosened and malposed teeth by the removal of tartar followed by the rearrangement of the teeth, so they could "function well" and would not suffer a "subsequent displacement." He utilized force, occlusal adjustment, interproximal stripping and wire ligation. Mills (1881) used ligatures of silk and wire, and elastics for tooth movement in the treatment of "Rigg's Disease." Apparently the methods of minor tooth movement in current use have long been known by periodontists.

A review of the literature reveals that full orthodontic therapy for adults has been practiced in a limited number of cases since

1854. Since these cases were treated for orthodontic reasons, the pre-treatment photographs, casts and roentgenograms show what appears to be good bone and soft tissue structure. These structures appear to be unchanged in the post treatment recordings. The age range of the reported cases is from 18 to beyond 50. Therefore chronologic age need not be regarded as a contraindication to treatment.

AREAS OF RESPONSIBILITY FOR PERIODONTIST AND ORTHODONTIST

Both orthodontist and periodontist are concerned with tooth movement, each from his therapeutic point of view. Mutual understanding and interchange of ideas would be beneficial.

The orthodontic point of view is concerned with physical abnormalities in the development, growth and establishment of occlusion. It includes the disarrangement of teeth, spatial disharmony of arches and structural disturbance of bone. Dentitions may be classified as normally occluded or malocclueded on the basis of tooth position (Angle, Lischer) or cephalometrics. The decision for treatment by the orthodontist is based both on classification and etiology, the aim being to correct a deviation from the defined norm. These deviations are apparent in the younger age population, with whom the orthodontist is usually concerned.

Periodontal cases usually are drawn from an older age population. The periodontic point of view is concerned with the existence of periodontal pathosis. The presence or absence of an underlying malocclusion is not, in itself, a significant factor. The decision to do minor tooth movement depends upon whether periodontal disease is present and complicated by malposed teeth. If, for example, periodontitis has caused teeth to loosen and wander, treatment is indicated, the aim being to restore tissues to health and migrated teeth to proper position. This decision to move teeth should be further explored to determine whether the patient's best interests would be served by full orthodontic treatment. In problems of this nature a closer rapprochement of the two specialties would seem to be desirable.

NEED FOR FURTHER EDUCATION

It would be advantageous to utilize the superior ability and technical knowledge of the orthodontist in the treatment of these cases. Frequently the orthodontist refuses cases of periodontal etiology because of the patient's age, the limited nature of the movement to be accomplished and the very fact that pathosis is present. So long as many orthodontists are reluctant to become involved in periodontal cases, their skill and resources remain unavailable. The need for further training in periodontics for orthodontists becomes obvious. Equally compelling is the need for further training in orthodontics for the periodontist so that he may be better equipped to treat his patients. While graduate programs in periodontics include courses in orthodontics, it would be advisable that the programs be intensified.

The movement of periodontally involved teeth is essentially orthodontic movement, regardless of the term, "minor tooth movement." This term is misleading since it infers easy or limited tooth movement which may be performed safely even though basic knowledge and training are lacking. Under no circumstance should a practitioner undertake minor tooth movement without training. Information is available through formal courses, the literature and lectures.

CONCEPTS OF OCCLUSION RELATED TO PERIODONTAL PATHOSIS

Because the problem of the malposed tooth focuses our attention on occlusion, it would be profitable at this point to reconsider the implications of basic concepts of occlusion. The presumption exists that good function and periodontal health are consequences of a normal occlusion. The corallary presumption is that malocclusion leads to periodontal disease. If these premises are accepted, then one can conclude that changing a malocclusion to a normal occlusion will prevent periodontal disease and

maintain health. These hypotheses, which so readily lend themselves to the justification of specific procedures involving the "correction" of occlusion, have never been validated. They rest on subjective criteria or may be arrived at through rationalization. There is a significant need for the accumulation of data to substantiate or refute these hypotheses.

Malocclusion and traumatic occlusion are frequently confused. Most authorities are agreed that traumatic occlusion means trauma from occlusion. This trauma is evidenced by pathology in the periodontium, hence periodontal traumatism is the more accurate term. Since the condition of the periodontium and not the arrangement of the teeth confirms the diagnosis of traumatic occlusion, it is possible to have a perfect occlusion which is traumatic and a gross malocclusion which is not traumatic.

It should be noted that "traumatic occlusion" does not initiate pocket formation, a symptom of periodontal disease. Once inflammation is present, however, the occlusal factor contributes further to pocket formation. Since inflammation is a consequence of local etiologic factors, periodontitis may occur even though the dentition is normal according to occlusal standards, and may not occur even when malocclusion is present.

PHYSIOLOGIC RESPONSE OF TISSUES TO FUNCTION

To all ends occlusion as portrayed by study models is an architectural concept and, as such, is static. Once function is introduced, any static concept of occlusion must be abandoned. Teeth do not remain in the same state, form and position throughout life. Teeth move in function as part of the dissipation of energy generated by the contraction of the mandibular musculature. Forces produced in the muscle cells move the mandible, cause tooth contact and are transmitted to the periodontium. These forces may compress enamel, dentin and bone, and may tense periodontal fibers. Strain patterns appear in all the hard structures. The connective tissue elements are remodeled in response to stress. This is all accomplished on cellular levels as a type of tissue regeneration or healing. Some authorities consider the tissue's inadequate response to function in this manner as the significant factor in "traumatic occlusion."

Abrasion is another consequence of function. Tooth substance and filling materials wear. They may wear on the buccal, lingual, proximal or occlusal. Different substances (enamel, dentin, gold, amalgam, acrylic and silicate) wear at different rates. Teeth apparently adapt to this abrasion by drifting mesially and occlusally. Relative positions of the teeth may remain the same, or deviations so mild as to be unnoticeable may occur. The rearrangement of bone and periodontal membrane are the mediators of this migratory adjustment. This entire interplay may occur without the development of periodontal pathosis.

TISSUE RESPONSE TO FORCE FACTORS

The response to force factors is physiologic when it consists of migration, and abrasion and the remodelling of tissues without the production of pathosis. The pathologic response, periodontal traumatism, is marked by the widening of the periodontal membrane, loss of adjacent alveolar bone, etc. The actions which give origin to the force factors are mastication, swallowing, bruxism and habits.

What emerges as significant in any consideration of occlusion is that the sole criterion for pathology caused by force is the biologic status of the periodontium and not the disharmonious arrangement of teeth.

When the biologic status of the periodontium is compromised by the presence of local inflammatory disease, as in periodontitis, does the response to force factors change? Insofar as healing of tissues is concerned, and trauma by definition constitutes wounding, there is a basic pattern for repair. Repair and reorganization attempt to follow the same pattern in health and disease. However, from the local point of view, inflammation has entered the picture

and will modify tissue reaction to force.

Firstly the amount of tissue fluid has increased and affected the physical consistency of the tissue. Inflammation produces edema, exudate, capillary proliferation and swelling. Thus the teeth may be loosened and physical resistance of the tissue must be altered. Following surgery the inflammatory response which is part of the healing phase often loosens teeth.

Secondly the production of granulation tissue and excessive fibrotic connective tissue is capable of exerting force. This force may move teeth out of alignment. These displaced teeth may return to their position when the granulation tissue is eliminated. A similar readjustment occurs when excess tissue is removed in Dilantin Hyperplasia.

The major consequence of inflammation is the destruction of bone and periodontal membrane which produces a change in architectural support. As a result of this grouping of changes, there is a difference in the *quantity* and *quality* of supporting tissues. It then becomes possible for antagonistic teeth to have differences in their supporting tissues in the course of the disease. The variations in support permit drifting and abrasion to occur in a non-uniform manner. This is further complicated by the fact that abrasion may occur differentially as a result of tooth hardness, form and position, or hardness of filling material. Surfaces worn unequally may receive and transmit forces unequally. Hence functional forces in the presence of inflammation may further loosen teeth and move them out of position.

When a tooth is moved out of position and the inflammation is resolved and the force factors become inactivated, the tooth tends to remain in that position. However, if the tooth is moved into contact on an inclined surface, the movement becomes self-perpetuating. The teeth tend to continue to move along the slope until an equilibrium is reached. When the occlusal situation precludes adaptation by abrasion or migration, then bony support will be lost, the tooth will be loosened and may even be exfoliated. The fate of the tooth depends on the biologic status of the periodontium and its response to force factors.

TISSUE RESPONSE TO MINOR TOOTH MOVEMENT

The tissues respond to orthodontic forces in the same manner as they respond to occlusal forces, namely by the remodelling of bone and connective tissue. Since the force applied and the occlusal relationships are controlled by the operator, situations producing pathologic response may be avoided. As Moyers points out, the movement occurs by virtue of the periodontium and not the appliance. Reitan, who has made a histologic study of tissue reactions to orthodontic force, shows that the adult periodontium is capable of supporting orthodontic movement.

Most sources are agreed that inflammation is a hindrance to orthodontic progress. When the inflammatory phase is controlled then the tissue may be considered normal in a cellular sense, despite the fact that the bone loss constitutes an abnormality in a structural sense. The pathology about such a tooth has been arrested and it is as suitable a candidate for movement as is a tooth without bone loss. When there is loss of bone movement occurs sooner since less reorganization of tissue is required. The center of rotation has been moved closer to the apex and small forces can produce greater movement and in less time. Because the movement is a matter of periodontal response, properly filled root canals present no problem.

Excessive force may cause necrosis and thus delay movement. Rear resorption of the necrotic tissue and the reestablishment of a normal tissue bed must occur before movement can proceed. Strong force is capable of producing trauma as evidenced by pain and loosening, and therefore minimal force is suggested in practice. Too rapid movement may injure the vascular bundle to the pulp and cause pulpal necrosis. Root

resorption is another consequence of excessive force.

Further pathology may be introduced by an appliance which is not stable and permits jiggling of teeth. The appliance moves the tooth in one direction while occlusal forces move the tooth in an opposite direction. Such oscillatory movement produces a widening of the periodontal membrane and a loss of adjacent alveolar bone. This constitutes periodontal traumatism.

ORDER OF TREATMENT PROCEDURES

Minor tooth movement follows the comprehensive examination and curettage. Surgery, occlusal adjustment and splinting procedures normally follow the completion of the movement. The sequence may be varied in accordance with the judgment of the operator.

As inflammation is resolved, granulation tissue removed and occlusal interferences cleared, the teeth may move back to position. When the teeth remain malposed minor tooth movement is necessary. It is desirable before proceeding to take new study casts since changes may have occurred since the institution of treatment. The patient, the casts and the roentgenograms should be restudied in an attempt to reconstruct the development of the malocclusion. Frequently such an understanding is helpful since treatment may involve the reversal of the pathways taken by the teeth in migrating.

CONSIDERATIONS IN TREATMENT

What are the standards that must be satisfied in selection of cases? Under what circumstances is minor tooth movement indicated? What therapeutic purposes are served? What precautions should the practitioner observe?

1. *The malposed teeth should be related to periodontal pathosis.* Malposed teeth with normal supporting structure are not normally treated by the periodontist. Conditions which are primarily developmental such as disturbances in the growth of the maxilla and mandible, gross disparity or malformation of arches, gross malpositions of segments of teeth and dento-facial anomolies are more properly in the province of orthodontics.

2. *The malposition must be of local etiology and must have no significant systemic relationships.* In the discussion of the biologic status of the periodontium, reference to systemic factors was intentionally omitted. Obviously systemic conditions may be related to periodontal disease. However, if the case history provides no suggestive leads and the clinical examination reveals no unusual findings, the implication of systemic background is unwarranted. Systemic conditions involving metabolic changes which may indirectly cause tooth migration or influence repair preclude therapeutic movement (e.g. Pagets, uncontrolled diabetes, hyperparathyroidism, etc.) unless the basic condition can be corrected.

3. *The malposition must be amenable to correction.* A prerequisite for therapy is adequate space. The movement must not be blocked by interfering cusps, since this would produce traumatism as well as interfere with movement. Desired space may be created by occlusal grinding, interproximal stripping, extraction or orthodontic movement of adjacent teeth. A bite opening appliance may provide occlusal clearance. Anchorage may be a problem if many teeth are missing or if there is a minimal bony supportive structure.

4. *The therapy should not be destructive.* The teeth should not be moved off basal bone, neither should they be moved so that the apices perforate the alveolus. Apices should not be tilted against adjacent roots, nor should the movement obliterate thin interproximal septa. It is inadvisable to attempt movement without first resolving inflammation. It is poor planning to proceed with movement when the final alignment would still be inadequate. If therapy would be a liability in terms of bone loss, minor movement is not permissible.

5. *Myofunctional factors must be adequately controlled.* When oral habits and muscular patterns have contributed to the malpositioning, we must first affect a correction before attempting minor tooth movement. Among the conditions we may deal with are tooth doodling, foreign object biting, clenching and clamping, bruxism, cheek and lip biting, tongue thrusting, exploring dental materials with the tongue and infantile patterns of swallowing. We may have to contend with muscle hypertonicity and hypotonicity and muscle length. This requires a knowledge of habit breaking principles and muscle physiotherapy. If the habits are not corrected then the final tooth positions will not be stable, and the teeth may drift.

6. *Operative and prosthetic needs may be fulfilled.* Movement may upright teeth so that they may be parallel, and also transmit occlusal forces in the direction of the long axis. Uprighting of teeth may prevent pulp exposure in operative procedures. Advantages such as better alignment with teeth in the same or opposing arches, more pontic space, wider embrasure opening, better cusp, fossa and marginal ridge relationship may be produced. Minor movement may minimize or avoid operative or prosthetic procedures. Uprighting may permit the use of fixed bridgework, rather than a removable partial. The closure of open contact points may eliminate the need for inlays for the same purpose. Intrusions and alignments will minimize grinding in occlusal adjustment and might thus prevent sensitivity or exposure. Movement of a grossly malposed tooth may prevent its extraction for prosthetic needs.

7. *Periodontal needs may be fulfilled.* There may be need to change situations which are contributing to periodontal breakdown. Some examples follow: (a) malpositions which cause food impaction; (b) irregularity and crowding may interfere with proper home care; (c) premature contacts on free-riding tooth slopes, producing loosening and drifting; (d) when tissue adjustment to traumatic contact is prevented by adjacent or occluding teeth. This situation is analagous to jiggling and is followed by similar loss of supporting structure; (e) when tissue is traumatized by tooth contact as may occur in deep overbite.

8. *The newly moved teeth must be retained in position with relative stability.* The resolution of inflammation and control of force factors have eliminated conditions conducive to migration and loosening. However the teeth may be loose following movement and some form of retention may be necessary. In those instances where splinting is part of the prescription there is no further problem. In other instances wire ligation is satisfactory solution. Occasionally night guards or Hawley appliances may serve for retention, but these can contribute to jiggling. If the teeth are not retained in position after movement they may drift and thus nullify the therapy.

9. *The satisfaction of esthetic needs.* The mental health value of esthetic correction is obvious, and yet the importance of orthodontic movements to satisfy esthetic needs is too often minimized. We are all familiar with the embarrassed patient who screens her teeth with her hand, or who draws her upper lip over protruding teeth. Correction of such conditions offers substantial mental hygienic benefits. The patient becomes increasingly upset over the implied prospect of losing teeth as periodontal symptoms become more obvious—recession, bleeding, looseness, migration and pain. With the realignment of teeth the patient is reassured and feels more secure. The comfort that accompanies the correction of anterior malpositions is a positive emotional feeling. Caution in case selection is indicated when the presenting patient confuses an imagined state of the teeth with the real situation.

UNDERTAKING TREATMENT

The decision to proceed with minor movement is a conclusion reached through the reexamination and the considerations,

as presented. The therapeutic aim is to counteract the etiologic factors that lead to the malpositioning; and by realignment of the teeth to produce a functional occlusion, and not necessarily a perfect one.

The treatment involves the application of force acting in the direction in which the teeth are to be moved. The force is applied by means of mechanical devices. The best device is the simplest one that will accomplish the movement without damage. Appliances are described in all standard orthodontic textbooks.

The appliances are implements to be used once diagnosis and prognosis are ascertained. The appliance does only what the perception and dexterity of the operator directs, influenced by the ability of the tissue to adjust and repair. If because of too ambitious a plan, excessive enthusiasm or lack of knowledge, the dentist overlooks personal, mechanical or biologic limitations, damage is likely to follow.

TEETH CONTINUE TO ADJUST FOLLOWING THERAPY

Once therapy is completed tooth position is not static. The teeth continue to respond to force factors and perhaps inflammation by loosening, drifting and abrading. It is best to view the repositioning as a shepherding of stray teeth so they can continue their adaptive movements together. These movements are generally overlooked in the consideration of orthodontic relapse. The adaptive juggling of tooth position in dynamic response to periodontal disease, oral force factors, reparative phenomena and possibly the anterior drift of teeth and mandible, may contribute significantly to relapse.

BENEFITS OF TREATMENT

Among the anticipated benefits of therapy are these:

(1) The possibility that properly aligned teeth may be able to make functional adjustments in health, prolonging the life span of the dentition.

(2) The possibility that through proper juxtaposition bone and tooth will better support adjacent bone and tooth.

(3) The patient will have enhanced pleasure, comfort and interest in the teeth.

(4) The patient's positive attitude may lead to a better maintenance of tissues by more diligent home care.

(5) The closure of diastemas may produce higher interdental septa. This has been observed clinically and may be explainable as the result of compressive force.

(6) The possibility that proper tooth position may through function protect or stimulate the gingiva, promoting tissue health.

CONCLUSION

The malposed tooth is not an isolated, static phenomenon. It is the result of adaptive responses which occur in an ever moving dentition. Each change arises in and leads to modifications in adjacent and antagonistic teeth. Since displacement is minute and the rate is slow, the teeth appear to be as immobile as are study casts. Comparison of casts over years reveals the migration and abrasion. The degree of response by migration, abrasion or looseness is probably interrelated.

An attempt has been made to introduce a broader base for the consideration of tooth malpositions of periodontal origin. Tooth wanderings may be terminal phases of periodontal disease, but they are indigenous to inflammation and trauma at every phase. Therapy may range from the closure of a central diastema for esthetic reasons, to the realignment of critical teeth and thus serve as the major determinant in the choice between periodontal prosthesis or full extraction.

It is hoped that this presentation will serve to stimulate further research and study, extend educational efforts and give rise to new insights and perceptions into an area of broad theoretical and clinical implication to the practice of dentistry. *630 West 168th St., New York, N. Y.*

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Reattachment—A Case in Point

by SAMUEL C. ROCKOFF, D.D.S.,* HERMAN S. ROCKOFF, D.D.S.,* AND ALVIN M. SACKLER, D.D.S.,† *Brooklyn, New York*

THE detachment of gingival tissue from hard tooth structure by the destruction of the periodontal membrane fibers and the apical migration of the epithelial attachment may create a periodontal pocket followed by loss of alveolar bone. Histologic reattachment is the restoration of the integrity of the connection between periodontal tissue and hard tooth structure. This phenomenom may be divided into two aspects: (1) the attachment of the epithelium at a more coronal level than the original base of the pocket, (2) the regeneration of new periodontal membrane fibers embedded in a new cementum and accompanied by a formation of new bone. This is to be distinguished from "clinical reattachment" which is in reality a close physical adaptation of the soft tissue about the clinical neck of the tooth.

Opinions regarding the likelihood of reattachment have ranged from definite "no's" to "doubtful," "possible," "probable," and "yes." Leonard[5,6] states that the epithelial lining of the pocket is capable of reattaching itself to the tooth providing the tooth surface is free of debris and compressed by the surrounding connective tissue. Box[2] reported a microscopic section representing this condition. Orban[9] claims that reattachment of the epithelium to the cementum would require the extension of protoplasmic processes from the basal layer of the epithelium into spaces created by the degeneration and loss of the periodontal membrane fibers. The surface of the epithelium approximating the root of the tooth is the superficial layer which contains no such protoplasmic processes. Attachment of this layer to the tooth would therefore be inconsistent with the histologic nature of the epithelial attachment. Glickman[3] notes that if the epithelial attachment and the epithelial lining of the pocket are

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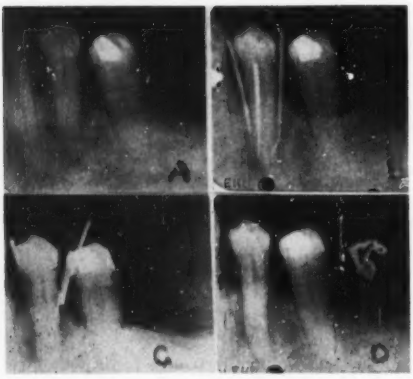

Fig. 1. Roentgenograms illustrating depth of pockets before and after treatment. A—2-13-53; B—2-13-53; C—4-11-53; D—7-31-53.

removed, and the root surface is thoroughly debrided of all degenerated and foreign material, some degree of "reattachment" would occur above the previous area of attachment of the epithelial attachment.

Goldman[4] and others state that, while reattachment of the gingiva and regeneration of bone is possible in the infrabony pocket, such is not the case in the gingival pocket. Clinical evidence of reattachment and regeneration in the infrabony type, supported by roentgenograms, has been reported many times in the literature. Beube[1] reported a case of reattachment in an infrabony pocket. This was supported by some histologic and radiographic evidence.

Failure to obtain reattachment in the treatment of periodontal pockets may be attributed to an environment not conducive to the repair process. More favorable local circumstances must be established through:

(1) debridement and elimination of the chronic inflammation.

(2) immobilization of the tooth and supporting tissues.

(3) protection of the operated area from destructive factors such as saliva, food, debris, stress, etc. to permit the reparative elements to organize.

In this study we have established the foregoing conditions and have sought to prove that histologic reattachment can take place.

Procedure: Because of prosthodontic considerations the lower right first premolar tooth of Mrs. M.L.,

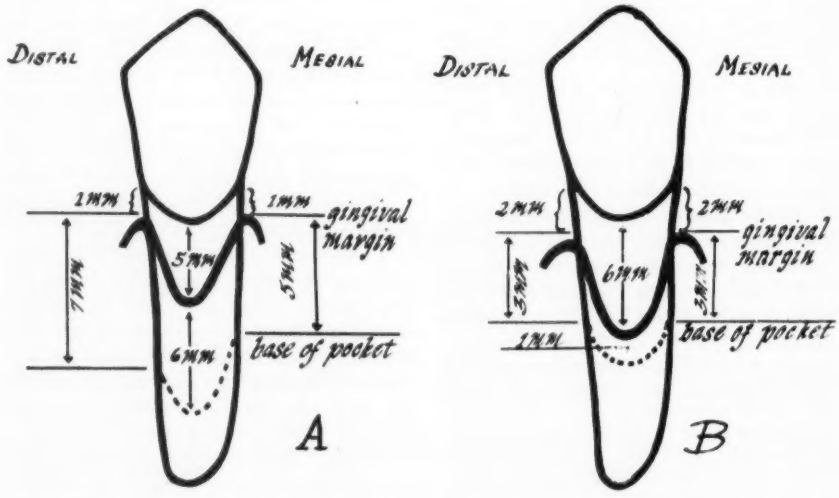

Fig. 2. Diagram showing pocket depths and location of gingival margins as found clinically before (A) and after (B).

Fig. 3. Splint and Surgical Pack in place, following operation.

age 56, was to be extracted. A roentgenogram (Figure 1) taken 2/13/53 shows great bone loss and film B gives some indication by means of gutta percha points of the depth of the periodontal pockets. Clinically, (Figure 2A) there was much recession of the gingival margin, and pockets of 5 to 7 mm on mesial, buccal and distal aspects, (shallow sulcus on lingual). There was much subgingival calculus and gingival inflammation, a grossly traumatic relationship with the opposing upper tooth, and extreme mobility (class III). The prognosis was apparently hopeless.

Before any specific treatment was attempted for this tooth, general conservative periodontal treatment was instituted to restore good oral hygiene and a healthy milieu.

On this specific tooth, the root surfaces were planed, the soft tissue curetted, the occlusion relieved, and an extracoronal acrylic splint was applied to stabilize the tooth through support by the adjacent teeth. After one week there was a decrease in the gingival inflammation but only a very slight change in the mobility and little change in the pocket depth.

On 2/20/53 under local anesthesia the following operation was performed. With a long thin tapering diamond stone, the pockets were entered and the epithelial attachment, the epithelial lining, the cementum, and even the alveolar bone crest were scarified. At the base of the pocket, the cementum and dentine were notched with an inverted cone bur. The area was debrided, a blood clot allowed to form, and dryfoil applied over the area. A surgical dressing was then placed over the foil, and the acrylic splint replaced (Figure 3). The whole area was left undisturbed for three weeks. On 3/13/53 the dressing was changed, the splint replaced and left for another week. Thereafter, the splint alone was worn for one month during which time the patient was taught to massage the gingival tissue gently.

Result: The soft tissue healed rapidly with a recession of the gingival margin of about 1 mm. and a remarkable reduction in pocket depth as shown in (Figure 2B). Roentgenograms taken on 4/11/53 with gutta percha points in position show shallowness of pockets (Figure 1C). Three months later the roentgenogram of 7/31/53 shows the filling in of bone (Figure 1D).

Fig. 4. Mesio-Distal section showing notch on Distal Aspect of Root at Base of original pocket.

On 8/28/53 under local anesthesia a block section including the tooth and periodontium of buccal and distal aspects was removed. Serial sections were made mesio-distally. Figures 4 to 8 illustrate the findings.

HISTOLOGIC STUDY

Histologic sections of the area above the notch, which was cut into the tooth during treatment (Figure 4) demonstrates that new periodontal tissue was formed. Evidence of new cementum is seen (Figure 5). Bone repair is demonstrated (Figure 5) at a point opposite the newly formed cementum.

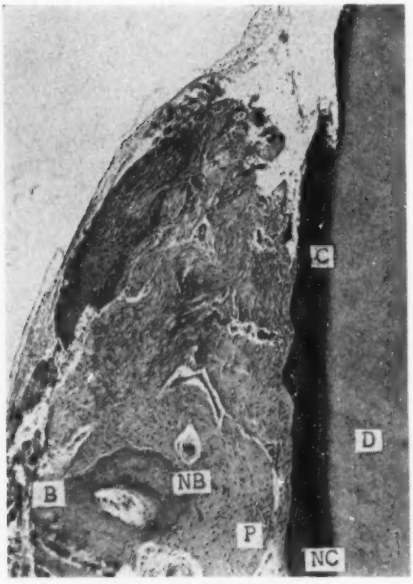

Fig. 5. Low Power View at Level of Alveolar Bone Crest and Gingival Margin. B, Bone; NB, New Bone; C, Cementum; NC, New Cementum; D, Dentine; P, Periodontal Membrane.

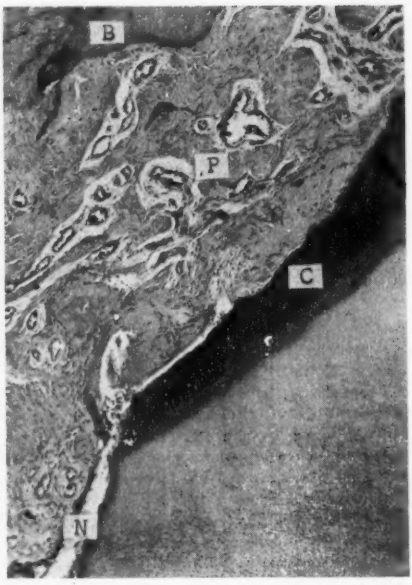

Fig. 6. Low Power View Just Coronal to Notch. N, Notch; C, Cementum; B, Bone; P, Periodontal Membrane.

The new periodontal membrane appears to be unorganized with most fibers arranged parallel to the long axis of the tooth (Figure 6, 7). This phenomenon is said to be characteristic of a regenerated periodontal membrane which has not yet been subjected to functional stresses. In a higher power (Figure 8), periodontal membrane fibers can be seen attached to the cementum.

As previously described, the periodontal pocket that was treated was located on the distal surface of the lower first premolar and extended around the buccal aspect to the mesial surface. This was confirmed by the insertion of the gutta percha points into the pockets and the roentgenograms prior to treatment (Figure 1). The roentgenograms A and B of 2/13/53 disclosed that two thirds of the root of the premolar were denuded of alveolar process and periodontal fibers due to a deep infrabony pocket. The roentgenogram of 4/10/53

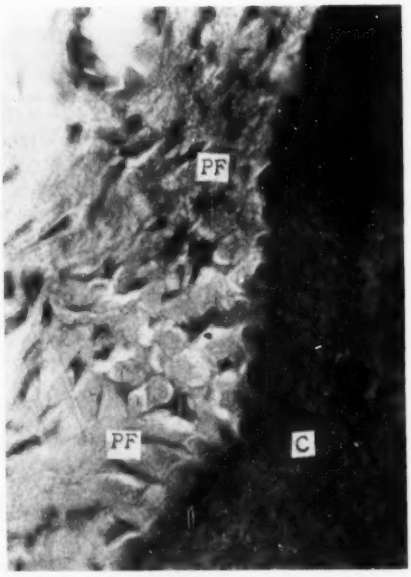

Fig. 8. O.I. View of Attached Periodontal Fibers. C, Cementum; PF, Periodontal Membrane Fibers.

Fig. 7. High Power View of Unorganized Periodontal Fibers. B, Bone; P, Periodontal Membrane; C, Cementum.

showed the bone filling in with the gutta percha points able to be inserted only a short distance into the pockets. The roentgenograms of 7/31/53 disclosed that the radiolucent area as seen at 2/13/53 was filled in with alveolar bone.

The microscopic evidence indicates attached unorganized periodontal membrane fibers, repairing bone, and new cementum, which constitute a regenerated supporting apparatus for the tooth. The sections do not show epithelial proliferation into the operated area. The attachment of the epithelium at a more coronal level than the new connective tissue was not demonstrable.

SUMMARY

This case is offered as evidence that histologic reattachment of the periodontal membrane can take place under certain optimal conditions. More cases will be needed to substantiate this finding and to help establish reattachment as a definite fact. Difficulties lie, however, in the acquisition of human experimental material. Only with a sufficient supply of such material, and a more advanced understanding of the cause of pocket formation in periodontal disease will it be possible to develop a standardized operative technique.

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Periodontosis in a Child with Hyperkeratosis Palmo-Plantaris

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THE very rare occurrence of the dermal lesion hyperkeratosis palmoplantaris in combination with periodontosis, as described first by the French authors Papillon-Lefèvre in 1924 and later amongst others by Rosenthal in 1951, gives rise to this article.

CASE REPORT

A.B., girl, 10 years old.

Shortly after birth the girl suffered from eczema in the face for some months. When 1½ year old, skin changes of hands and feet appeared: the skin in these areas became coarse and scaling, some superficial grooves were noted. In the beginning the skin changes intensified and now sometimes, especially in summer, the grooves tend to deepen and to become a nuisance. At other times the skin changes were said to disappear almost completely.

The deciduous teeth erupted normally, but shortly afterwards the gums became inflamed and purulent; the teeth loosened and caused much trouble while eating. The teeth were lost, so at the fourth year of life only one molar was left; 1½ year later this molar was lost too. Her permanent teeth erupted normally, but at her 7th year of life she suffered again from inflamed and suppurating gums. This inflammation is still present; otherwise she enjoys good health and has never suffered from special diseases.

She is an obese girl, weighing 37.5 kg when 6½ years old. The tractus urogenitalis, cerebrospinalis, digestivus, respiratorius and circulatorius function normally.

Family history: nothing abnormal, no other examples of identical or analogue skin or mouth changes have occurred. Her father and mother are first cousins.

General examination. Obese girl; weight 46.7 kg, length 1.34 m; hair normal. Head: brown eyes; nose and ears: nothing abnormal. Mouth: tongue and oral mucosa (the gums excepted) normal.

Present Status of Teeth

Depth gingival sulcus, (buccal)	1½			1½	1½	3	3							2	1½	1½	1			8	2mm
Teeth		6			4	3		2	1					1	2		3		4			6	7	
present		6	5			3		2	1					1	2		3		4	5	6	7		
Depth gingival sulcus, (buccal)	8		2			5		9	10					7	7		2		2	2	5	2mm		

Normal anatomic form of teeth; for nonerupted teeth see X-ray examination. *Upper jaw:* diastema between lateral and central incisors; insufficient space for eruption of $\underline{P_2}$; $\underline{|P_2}$ erupts at the palatinal side. *Lower jaw:* Diastema between $\underline{C|}$ and $\underline{|_1}$, diastema between $\underline{|_2}$ and $\underline{|C}$. The shape of the jaws is normal. *Caries:* occlusal caries in first molars; some temporary fillings. *Gums:* (fig. 1) marked glossy marginal tumefaction, soft consistence and easy bleeding of both free and attached gingiva at the buccal and lingual resp. palatinal sides.

Pockets: depths given in the diagram above; on palpation pus appears.

Further abnormalities: sordes; foetor ex ore; supra- and sub-gingival calculus.

Mobility of teeth:			4	3	2	1		1	2	3	4		6	7
firm	5		3							3	4	5		7

All teeth were firm, except

some mobility $\underline{6|}$

movable: max. displacement < 1 mm $\underline{6\ 2|\ \ 6}$

movable: max. displacement > 1 mm $\underline{\ \ 1\ |\ 1\ 2}$

University of Utrecht, Sub-division Periodontology, School of Dentistry; Department of Dermatology, University Hospital.

Fig. 1. Periodontosis in the 10 year old child.

The tonsils are somewhat enlarged; Glandula thyroidea and trachea: normal; Lymphoglandulae: some enlarged submandibular lymphnodes; Heart and lungs: nothing abnormal. Bloodpressure: 110/65. Abdomen: normal, liver and spleen not palpable. Extremities: on the right leg positive Babinsky, other reflexes normal.

Skin changes: the skin of the palms (fig. 2) is red, thickened; some scaling is present. The changes are rather sharply demarcated reaching as far as 1 cm on the wrists and till halfway at the sides of the fingers and covering the thenar. The interdigital spaces are completely free; except those between dig. I and II. The design of the skin is accentuated; some superficial fissures are present. The soles (fig. 3) are more markedly affected by these changes, especially the edges of the feet and the heels. The eruption extends till over the medial and lateral aspects of the feet. It involves the medial surface of the big toe and spreads over an area of about 4 cm above the heels. The flexor aspects of the big toes are slightly red and show some superficial fissures. The other toes and all interdigital spaces remain unaffected. The malleoli are not involved. A marked degree of hyperhidrosis of palms and soles was noted. Otherwise the skin presents a normal aspect.

At both legs just infrapatellar a red, slightly scaling, area is present.

Pathology: Biopsy of the plantar area of the right foot (fig. 4). Epidermis: the stratum corneum is slightly thickened. In many areas the cells contain their nucleus up to the surface (parakeratosis). The height of the stratum granulosum varies considerably, from up 2 to 7 cells. This layer is mostly narrowed at the nucleus containing areas of the horny layer; but is nowhere absent. The stratum nucosum appears normal.

Cutis: nothing abnormal.

Laboratory: Blood: sedimentation rate 18-40 mm. Hb 85%; erythr. 4.8.00.000; leucoc. 6900; diff.: eos 1, rods 7, polynucl. 55, lymph. 34, mon. 3%; vitamine A 9 I.U./10 ml serum (normal), urea 31 mg %. The bloodsugar curve was slightly flattened. Liverfunction: thymoltest normal; alkal.fosfatase 15 U. Urine: nothing abnormal, 17 ketosteroid excretion normal. Pirquet reaction: bov. –, hum. –. Basal metabolic rate: – 9%.

Fig. 3. Plantar changes.

Fig. 2. Palmar changes.

X-ray examination: Chest: lobus venae azygos; no other abnormalities. Skull: thin calciumline* located in attachment of the tentorium (?). Hands and feet: normal bone-development. Teeth (fig. 5): processus alveolaris mandibuli: diffuse resorption of bone in the frontal area and at $\underline{6}\,|\,\underline{6}$; processus alveolaris maxillae: horizontal resorption of bone in the region of the front incisors.

The following teeth which have not yet erupted are present and seem normal:

$$\begin{array}{c|c} 7\ 5 & 5 \\ \hline 8\ 7\ 4 & 8 \end{array}$$

*Corson's case also showed intracranial calcium deposits.

Cases from literature

Year of publication	*Authors*	*Patients**	*Consanguinity of parents*
1924	*Papillon—Lefèvre*	brother and sister	first cousins
1927	*Petges, Delguel*	M	?
1938	*Wannenmacher*	M	?
1939	*Corson (Rosenthal, 1951)*	brother and sister	?
1941	*Woods, Wallace*	M	?
1942	*Wannenmacher*	M	first cousins
1945	*Jusem***	2 sisters	?
1951	*Plathner*	F	?
1952	*Bataille, Duperrat (Bataille, 1953)*	M	blood relations
1952	*Ionata*	M	?
1953	*Boisramé*	2 sisters	—
1953	*Köhler*	2 brothers	?
1955	*Duperrat, Monfort*	M	?

*The age of the patients varies between 2 and 23 years.

**In 1948 *Jusem* published another case, but did not mention the condition of the skin.

Bacteriology: pus culture from the pockets yields a streptococcus viridans sensitive to penicillin, streptomycin and aureomycin.

DISCUSSION

The first description of the combination of an inheritable form of hyperkeratosis palmo-plantaris combined with periodontosis was given by Papillon-Lefèvre in 1924, who believed the skin alteration to be the same as in Meleda's disease and thought the skin as well as the dental alterations to be the result of a disorder in the internal secretion.

a. *Meleda's disease*

Meleda (Mljet) is an island in the Adriatic near Dubrovnik. The first report about the occurrence of an heritable skin disease here dates from 1826 (*Stulli*, see *Hovorka* and *Eblers*). Afterwards the disease was studied by *Hovorka, Eblers, Neuman, Bošnjaković, Kogoj* and *Hoede*. The result of their reports shows that this disorder is inherited as an autosomal recessive. The disorder appears frequently at Meleda. At the present time about 15 of the (±) 2000 inhabitants of Mljet suffer from this disease (*Dörken*), because of intermarriages, as 15% of all marriages in this small community are consanguineous (*Bošnjaković*).

The report by *Uimanov* (see *Moncorps*), who remarked that the disease became rarer in the Mohammedans after intermarriages had been forbidden, also speaks for inheritance as a recessive. The first symptoms at hands and feet are present already at birth (*Kogoj, Hovorka, Eblers*). After some years the hyperkeratosis increases and gives rise to a thick hornlayer on palms and soles and at the flexural aspects of fingers and toes.

In these hyperkeratotic areas deep fissures develop, occasionally penetrating into the derma. The hyperkeratosis spreads slowly from the volar side to the dorsum of fingers and hands and over the wrists to the distal part of the underarms and in the same way to the dorsum of toes and feet, to the malleoli and the distal parts of the legs. In about 50% of all cases the hyperkeratotic plaques also occur at the knees and elbows. Thus the clinical picture may vary considerably. A marked hyperhidrosis with foetor of palms and soles is found in almost all cases. It is usual that with the advancing age the hyperkeratosis progresses.

b. *Hyperkeratosis palmo-plantaris combined with periodontosis. (Papillon-Lefèvre)*

As mentioned above, Papillon, Lefèvre described in their publication 2 patients (brother and sister) both suffering from "kératodermie palmaire et plantaire symétrique familiale (Maladie de Méléda) chez le frère et la soeur, coexistence dans les deux cas 'd altérations dentaires graves." The palms showed a sharply limited hyperkeratosis, which extended till the edges of the hands and the arms. The soles

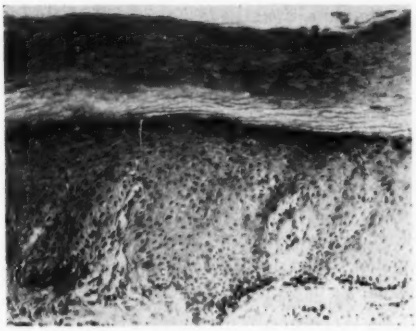

Fig. 4. Parakeratotic area in plantar hyperkeratosis.

showed the same anomaly, extending till over the edges of the feet, the Achilles tendon and the malleoli externi. Moreover red, somewhat scaling nummular zones were present at the tuberositates of the tibiae and in the brother also small areas of hyperkeratosis existed at the dorsal surface of the small toe and some fingerjoints. The skin at the elbows showed a normal aspect. The hyperkeratosis was not very marked, the skin was more parchment-like and of an accentuated design. Some scaling of the somewhat red, thin (?) skin was present. The changes in the skin were not bordered by a red area; the nails and hairs were normal; hyperhidrosis of hands and feet was noted.

Very conspicuous were the alterations of the teeth of which both patients suffered: the 4½ year old girl had lost 8 of the 20 deciduous teeth. Many teeth showed caries, while in other aspects the alterations were similar to "pyorrhée alvéolodentaire," showing loose teeth and retraction of the swollen gums at the cuspidates. The 6 year old boy had no teeth left since 2 years already. The upper and lower processus alveolares were resorbed to a marked degree; the upper jaw arch fitted in the lower one. The retraction of the upper lips gave the child the appearance of a toothless old man. Submaxillary some enlarged lymphglands were palpable.

The skin changes were believed to have developed in the third year of life. About this time also the first deciduous teeth were lost.

c. Other forms of hyperkeratosis palmo-plantaris do exist such as hyperkeratosis palmo-plantaris Unna-Thost inherited in a dominant. However also in these patients never the above mentioned periodontosis was noted.

Except a light degree of rhachitis and some pyoderma no other abnormalities were seen.

The 5 other children of the family were normal. Neither the parents nor the other members of the family suffered from similar anomalies. The parents were bloodrelations (first cousins).

In 1939 in dermatological literature a second case was published about this combination of symptoms (Corson) and in 1952 a third one (Bataille, Duperrat). In this latter case the parents were cousins too. In dental literature a number of publications have made this clinical picture better known. Perhaps some cases of juvenile periodontosis belong to this category (i.e. Lenstrup, Brinch and Koppen). The skin alterations may have been overlooked by the dentists, just as the dental changes by the dermatologists.

In the described cases the dental history is always of the same pattern:

After the appearance of the deciduous

Fig. 5. Diffuse advanced alveolar bone resorption. Lamina dura not present in most regions. Date: March 1955.

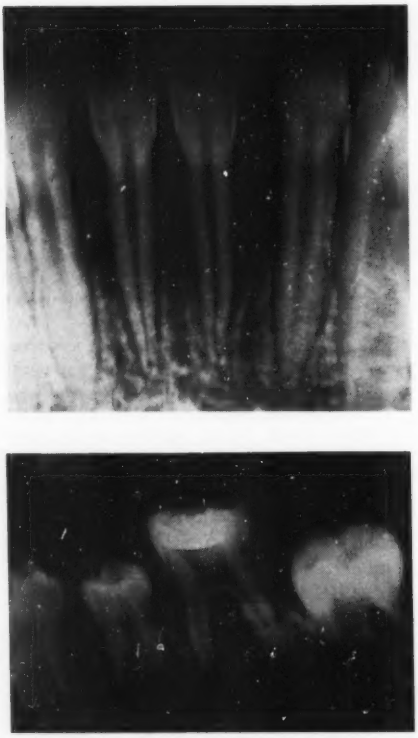

Fig. 6 a, b. Lower incisor region (a) and mandibular molar region, left side, showing bone resorption. Date: March 1954; Compare with X-pictures of one year before (March 1954), Fig. 5.

teeth a periodontosis develops at the age of about 2 or 3 years. The dystrophia of the processus alveolaris combined with inflammation of the gums give rise to the formation of pockets and the loosening and ultimate loss of the teeth. The suppuration is combined with foetor ex ore. During the 4th and 5th year of age all teeth are lost successively. Hereafter the symptoms of inflammation of the gums disappear. Nothing abnormal in the eruption of the permanent teeth has been observed, however shortly after their eruption the periodontal symptoms start again: pathological deepening of the sulcus gingivalis, suppuration and loosening of the teeth.

Sometimes all teeth may get lost; usually in the same sequence as their time of eruption. Most cases have been studied under the 12th year of life, therefore we are less informed on the course of the periodontosis of the permanent denture.

Wannenmacher and Harndt describe the loss of all first permanent molars and the mandibular central and lateral incisors before the age of 10½. Petges, Delguel mention in their case the loss of 15 teeth between the age of 10 and 13 years.

In Plathner's case on the 11th year no teeth had been lost yet. One patient of Rosenthal had a full upper and lower denture at the age of 16, the other one had a partial prothesis at her 12th year of life. In the 19 year old patient of Duperrat and Monfort only 4 molars were still present.

CONCLUSION

We must distinguish the following groups:

a. hyperkeratosis palmo-plantaris heriditaria progrediens (Meleda's disease); inherited as a recessive, b. hyperkeratosis palmo-plantaris combined with periodontosis (Papillon-Lefèvre), inherited as a recessive and c. hyperkeratosis palmo-plantaris (Unna-Thost); inherited as a dominant. No sex-limitation has been noted in these groups.

In dermatological literature Meleda's disease has repeatedly been diagnosed incorrectly. The diagnose ought to be restricted to those cases in which exist:

(a) excentric "glove-like" expansion of the hyperkeratosis palmo-plantaris, in a great number of cases even involving the distal parts of the extremities and (or) elbows and knees.

(b) a marked degree of hyperkeratosis (usually with deep fissures).

(c) transmission as a recessive

We believe it not to be justified to consider the hyperkeratosis palmo-plantaris described by Papillon-Lefèvre as a form of

Meleda's disease; in this regard we share completely the opinion of Bellenger. From none of the descriptions of the hyperkeratosis type Papillon-Lefèvre does the for Meleda's disease so typical excentric expansion of the hyperkeratosis appear. Another important argument against identity is the generally only very light degree of the hyperkeratosis noted in the entity described by Papillon-Lefèvre. The argument that the patients were still too young to judge this, is not valid as we know that in a 23 year old patient (Rosenthal), the same light degree of hyperkeratosis still existed. Moreover mouth-changes have never been noted in cases of Meleda's disease. Both disorders are inhired as a recessive.

We believe that the rare occurrence of the periodontosis with hyperkeratosis palmo-plantaris Papillon-Lefèvre justifies the description of a new case of this disease. We did not succeed to convince the child's parents of the usefulness of dental treatment. The destruction of the alveolar bone proceeds in a very high tempo, as can be noted by comparison of the X-status of March 1955 (fig. 5) with the X-pictures of $\overline{2\ 1}\ |\ 1\ 2$ and $\lceil\ 6$, made just one year before (March 1954) (fig. 6a, b).

SUMMARY

Description of Periodontosis in a child with Hyperkeratosis palmo-plantaris (Papillon-Lefèvre); this entity is compared with Meleda's disease. A new case of hyperkeratosis P.-L. is added. The anomaly is inherited as a recessive. The authors are of the opinion that this type of hyperkeratosis palmo-plantaris combined with periodontosis ought to be considered as an entity which is different from Meleda's disease.

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The Infrabony Pocket: Classification and Treatment†

by HENRY M. GOLDMAN, D.M.D.* AND D. WALTER COHEN, D.D.S.**

IN recent years, the principal clinical lesion of periodontal disease, the pocket, has been studied clinically, radiographically, and histopathologically. As a result of these investigations it became apparent that the pocket had to be classified on the basis of the location of the bottom of the pocket in its relationship to the alveolar crest. Arising from these studies came the classification of pockets: (1) suprabony or supracrestal and (2) infrabony or subcrestal. The suprabony pocket is defined as a pathological sulcus where the base of the pocket is coronal or occlusal to the alveolar crest, while the infrabony is defined as a pathological sulcus where the bottom of the pocket is apical to the alveolar crest. The suprabony pocket was further subdivided into the gingival or pseudopocket and the periodontal pocket. This classification had merit not only from a teaching standpoint but also on a therapeutic basis.

Much attention has been focused on the infrabony type of pocket in recent publications and this lesion has been described as amenable to either the new attachment procedure or osseous surgery for its eradication. It became obvious to us from our observations of clinical as well as human skull material that a classification of the infrabony pocket was necessary not only for academic purposes but also to serve as a rational basis for the selection of a method of treatment.

The proposed classification of the infrabony pocket is on a morphologic basis and is dependent on the location and number of osseous walls remaining about the pocket. Much of this material studied was from human skulls where the gingivae and other soft tissues were intact. The location of the bottom of the pocket was established, the material radiographed, and then the soft tissue was removed. The remainder of the material was taken from clinical cases under treatment.

The first group of infrabony pockets described have three osseous walls. These trough-like defects are commonly observed in the interdental areas where one finds an intact proximal wall as well as the buccal and lingual walls of the alveolar process. Some of these lesions may be shallow with a broad orifice to the osseous part of the pocket while others may be narrow and deep. Three wall infrabony pockets are occasionally observed on the lingual surfaces of maxillary and mandibular teeth where the lingual plate is intact as well as both proximal walls. Less frequently noted are infrabony pockets located on the buccal surfaces of maxillary and mandibular posterior teeth. It is not uncommon to find them extending around the tooth to involve 2 or sometimes 3 surfaces. When the infrabony pocket is circumferential and involves the four surfaces of the tooth, it actually has four osseous walls (buccal, lingual, mesial, distal). This occurs infrequently.

The determination of the position as well as the number of osseous walls is of concern to the clinician during his examination procedures.

The radiograph can be of great aid in demonstrating the presence of buccal and lingual and proximal walls in a pocket occurring in the interdental area. Placing a radiopaque object such as a gutta percha point, a periodontal probe, or Hirschfeld

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point will enable one to locate the bottom of the pocket. Many times the buccal and lingual walls will register on the radiograph and one has to relate the radiographic observations with the clinical picture. Taking a probe and running it buccally and lingually one will engage either soft tissue or the osseous walls. If there is a doubt a flap can be laid back to see the outline of the pocket or a fine straight needle can be passed through the buccal and lingual soft tissue to map out the height of the crests.

Two wall infrabony pockets may be seen in the interdental areas. If the buccal and lingual walls are intact, but the proximal wall has been destroyed, the lesion is commonly referred to as an intraosseous interproximal crater. Because the base of the pocket is apical to either the buccal or lingual wall, this falls into the classification as an infrabony pocket. One may find that the two walls remaining are the buccal wall and the proximal wall or the lingual wall and the proximal wall. In these situations there is usually just a curtain of soft tissue remaining on the surface where the osseous wall has been destroyed.

One of the interesting aspects of the skull material is that when the soft tissue has been stripped out of the bony defect, the osseous surface may be made of cancelleous supporting bone especially in the interproximal wall. Sometimes a more dense sclerotic wall of bone was noted underneath the soft tissue. This latter situation usually appeared more radioopaque on the radiograph.

The infrabony pocket which has one osseous wall remaining is usually seen in the interdental area. Here it is most common to observe the presence of a proximal wall with the buccal and lingual walls destroyed. This can be detected clinically by probing or passing a needle through the soft tissue; radiographic examination may also be helpful. It is much less common to find the buccal wall intact with loss of the proximal and lingual walls or to have a lingual wall intact with the loss of the proximal and buccal.

In setting up a classification of this sort we realized that many of these infrabony pockets were not pure examples of either one, two, or three walls. It was quite common to find various combinations of osseous walls. We frequently observed areas where the apical part of the pocket had more walls than the coronal part. For example, we frequently noted a pocket where the bottom part of the pocket had 3 walls while in the coronal part the buccal and lingual walls were destroyed leaving just the one proximal wall. There are also pockets with 3 osseous walls at the apical part

INFRABONY POCKET

THREE OSSEOUS WALLS

a. proximal, buccal & lingual walls
b. buccal, mesial & distal walls
c. lingual, mesial & distal walls

Four osseous walls– buccal, lingual, mesial & distal

TWO OSSEOUS WALLS

a. buccal & lingual (crater) walls
b. buccal & proximal walls
c. lingual & proximal walls

ONE OSSEOUS WALL

a. proximal wall
b. buccal wall
c. lingual wall

COMBINATION

a. 3 walls + 2 walls
b. 3 walls + 2 walls + 1 wall
c. 3 walls + 1 wall
d. 2 walls + 1 wall

Fig. 1. Classification of infrabony pockets.

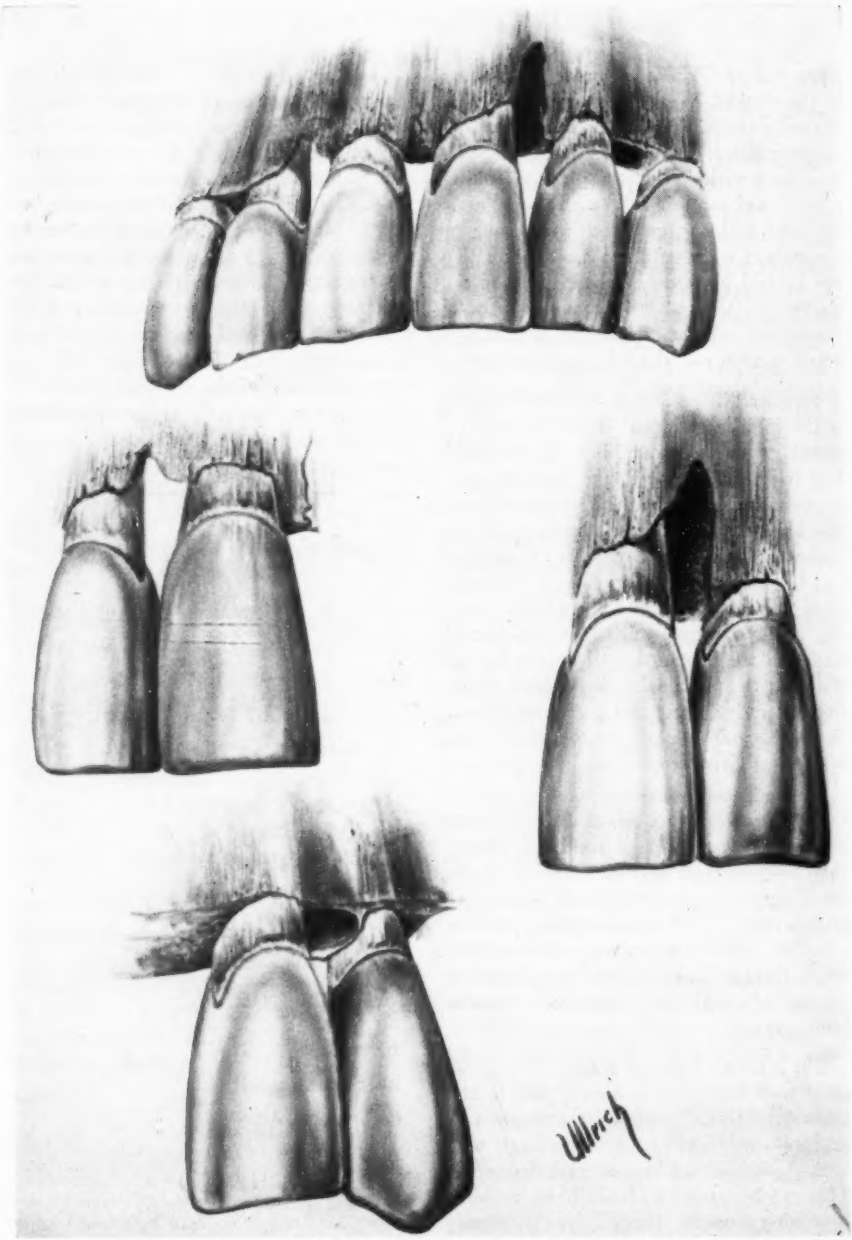

Fig. 2. Drawing of the various bony topographies associated with infrabony pockets. In the upper drawing, on the distal aspect of the left lateral incisor, a bony defect can be seen. This defect has osseous walls on the lingual, labial, and proximal; the latter serves as the mesial housing of the cuspid. This is classified as a three-walled infrabony pocket when the soft tissue is present. Distal to the left central incisor, the osseous housing of a two-walled infrabony pocket can be seen. The labial bone has been destroyed; the palatal and proximal walls remain intact. Mesial to the right lateral incisor the bony topography of a one-walled infrabony pocket is shown. Note that both the labial and lingual walls have been destroyed, with only the proximal wall remaining. In the center of the photograph on the right, the two-walled defect is shown in higher power, while on the left, the one-walled is illustrated. In the lower portion of the photograph, three bony walls may be seen.

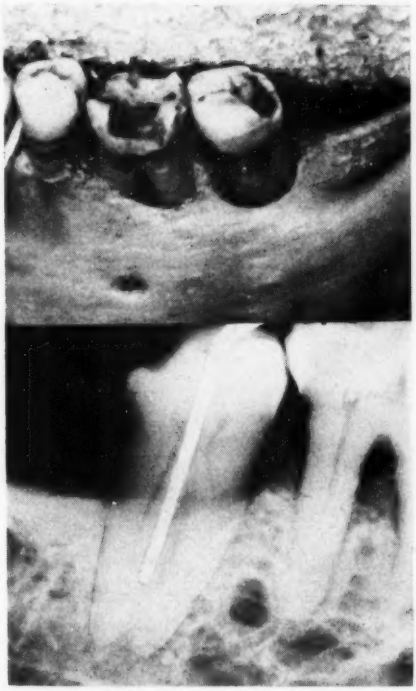

Fig. 3. Photograph of a skull showing a bony defect on the buccal aspect of the second molar associated with a three-walled infrabony pocket. In the radiograph a probe has been inserted into the bony defect. Note that the buccal osseous wall does not show up in the radiograph and only the base of the bony defect can be determined.

and 2 walls as the cemento-enamel junction is approached. We have also encountered situations in which there were two walls at the apical portion with one wall coronally.

This classification has served as a basis for our selection of therapy since we realize today that all infrabony pockets cannot be eliminated by means of new attachment, but one must employ other procedures, as will be brought out subsequently. This determination of which method is to be employed will usually depend on the topography of the lesion.

CLINICAL EVALUATION

One of the important aspects in the treatment of the infrabony pocket is the visualization of the topography of the pocket. The outermost limits of the detached gingival tissue must be explored. Since disease affects the entire circumference of the tooth in question, therapy must be directed toward healing this tissue. Also that portion below the bone crest must be outlined in respect to the surfaces of the tooth. At times this latter defect is confined to a single surface (most usually the proximal aspect) but many times is tortuous in nature, the deepest point being located on a different surface of the tooth than the orifice of the main portion of the pocket. Even in instances where a single surface pocket is encountered, small lateral pouches may be found. Not infrequently, what is thought to be an infrabony pocket on the mesial aspect of a maxillary anterior tooth, after probing is found to involve the palatal aspect as well.

Thus, infrabony pockets assume the shape of various combinations of short-and-deep and narrow-and-broad defects. They may be located on a single surface of a tooth or may encroach upon adjacent surfaces. Not uncommonly, one may find an infrabony pocket which is characterized by a narrow orifice, but at the base the defect is bulbous in form causing the pocket to resemble a bottle.

Radiographic examination of the infrabony pocket discloses a vertical resorptive lesion but gives no information concerning the base of the pocket. Actually, therefore,

Fig. 4. A three-walled osseous housing of an infrabony pocket on the palatal aspect of a maxillary molar may be seen. Note the destruction of the distal and mesial proximal walls as well as the palatal, resulting in a trough extending around the tooth.

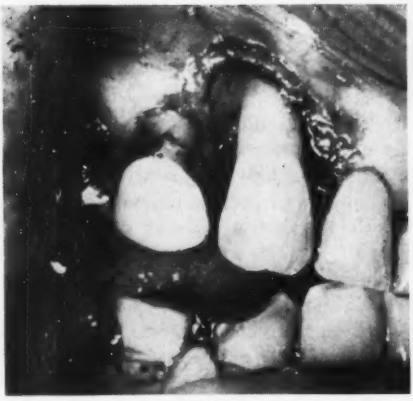

Fig. 5. The clinical photograph and radiographs of a two-walled infrabony pocket on the distal of the maxillary cuspid. Note that once the gingival tissue has been removed the bone exists only on the palatal and proximal aspects, the entire labial wall having been destroyed. Radiographically, there is a vertical resorptive lesion. One cannot determine from this radiograph whether the pocket is one, two, or three-walled. Only by clinical examination can this be ascertained. Passing a needle through the gingiva from buccal to palatal, and palatal to buccal, will give the information desired. Entering from the buccal aspect, if the needle cannot penetrate any distance, then a buccal wall exists. If it passes through then no wall exists at all. If it passes completely through to the outer surface on the palatal aspect, both walls are missing. Thus, in reversing the penetration, one can tell exactly what walls are present.

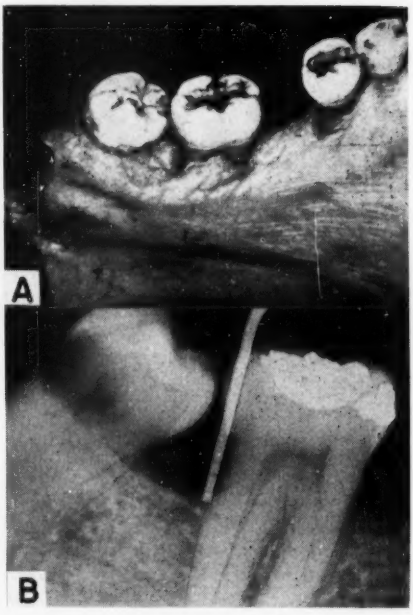

Fig. 6. Photograph of skull material and its radiograph. Note that on the distal aspect of the second molar there is a crater-like defect. However, the defect slopes towards the distal of the second molar and here the destruction takes on a broad resorbed defect. Radiographically, with the probe in position, one notes that in this area the bony housing is below that of the proximal wall. This may be classified as a broad, one-walled infrabony pocket defect.

one cannot recognize by radiology the presence of a pocket at all, for a vertical lesion may be present but the gingival tissue may be intact with no pocket formation. Radiographs taken after opaque materials have been inserted into the gingival retracted area (pocket) disclose the depth and contour of the pocket in respect to the bone outline. Unfortunately, this is one plane and a three dimensional picture cannot be visualized from this examination. Yet a certain amount of information can be obtained. The radioopaque materials are Hirschfeld points, gutta percha probes, and bismuth solutions impregnated in cotton.

Important, therefore, is the recording of the size and general topography of the in-

Fig. 7. Photograph and radiograph of skull material showing a mesially tilted third molar. Note that there is a broad resorptive trough on the mesial aspect of the tooth. This represents the bony defect associated with a one-walled infrabony pocket.

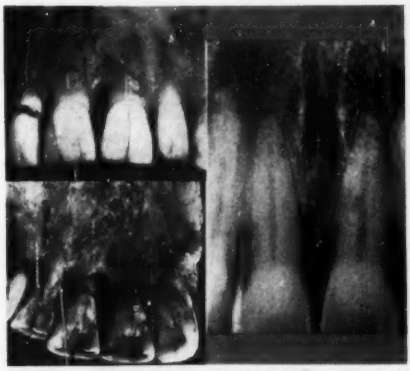

Fig. 8. The photograph and radiograph of a one-walled infrabony pocket on the mesial aspect of a maxillary left central incisor. One can visualize the bony defect associated with a one-walled infrabony pocket.

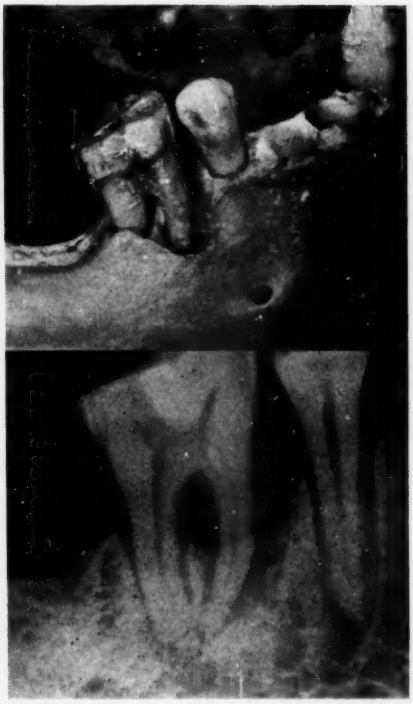

Fig. 9. Photograph and radiograph of skull material showing a combination of various topographies. At the base of the bony defect three walls are evident. More occlusally, two walls are present, while at the occlusal level, one wall, that of the proximal, remains. This type of defect must be carefully determined in treatment. The exact topography of the lesion must be visualized prior to the operative procedure for such a combination of walls would necessarily change the mode of treatment from that of a three-walled infrabony pocket. Clinical examination, and radiographic examination with radioopaque material, are necessary for the diagnosis. (Courtesy of Dr. John Prichard)

frabony pocket, especially that part submerged below the crest of the alveolar process. This consideration refers to the soft tissue portion of this periodontal lesion. Even more important, however, is the topography of the bony housing. Although radiographically the defect has been described as a vertical resorptive lesion, not always can information concerning the buccal-lingual walls be derived therefrom.

Infrabony pockets are seen in which the defect is bordered by three walls although more than one surface of the tooth is involved. This is often found on the palatal-

Fig. 10. Photomicrograph of an infrabony pocket on the mesial aspect of a maxillary cuspid. Seen in the photomicrograph is the lateral incisor on the left and the interdental septum and the cuspid on the right. Covering the interdental septum is gingival tissue. The base of the pocket is below the interdental crest of the lateral incisor. Note the pocket depth on the lateral incisor is extremely shallow. This photomicrograph was taken from a specimen with three walls.

Fig. 11. Photomicrograph of an infrabony pocket on the mesial aspect of a maxillary tilted molar. Note the distance between the tooth and bone. This photomicrograph was taken from a case with a two-walled infrabony pocket, the buccal wall being mesial.

proximal aspect of the maxillary teeth. Although radiographic examination is an aid in determination of the outline of the bony walls especially if radioopaque materials are inserted into the pockets before the radiographs are taken, it by no means gives accurate information. It is necessary that the clinical examination disclose the outline of the bone crest in relation to the base of the pocket. Probing in the pocket reveals the point to which detachment has occurred in relationship to the tooth. Therefore, in addition, the operator must probe by a sharp instrument (explorer or

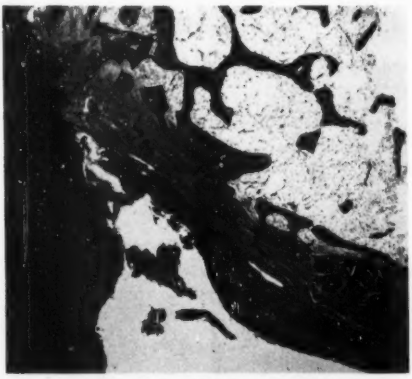

Fig. 12. This is a photomicrograph of an infrabony pocket on the mesial aspect of a tilted maxillary molar. In this instance the specimen contained only a proximal wall, the buccal and palatal being missing. Because of the wide distance between tooth and bone, this may be classified as a broad one-walled infrabony pocket.

Fig. 13. Clinical examinations of infrabony pockets are essential in diagnosis. The various topographies that may be present can be determined fully by clinical probing. Radiographic examination, while informative, is not in itself conclusive. Probing, from the buccal and lingual aspects, to determine whether these walls are present, is necessary. Also important is the determination of the location of the crestal portions of the walls that may be present.

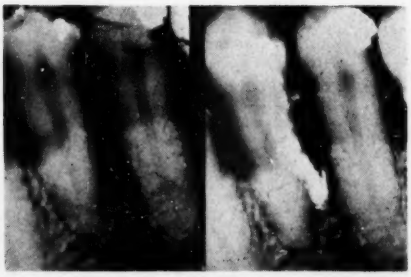

Fig. 14. Radiographs showing the base of an infrabony pocket. Associated with the infrabony pocket is a vertical resorptive lesion of the bone seen radiographically. One cannot determine from this whether the buccal and lingual housing is present. Hence, clinical examination is necessary. Also, one cannot determine where the epithelial attachment, locating the base of the pocket, exists. By inserting a radioopaque material into the pocket prior to the taking of the radiograph, this factor can be established. Note that in this instance the base of the pocket coincides with the base of the bony defect.

some sort of needle) for the location of the crest of the alveolar process. Should, for example, a proximal infrabony pocket be examined, one can pass a needle bucco-lingual from one side and then to the other, to determine whether these walls are present. Thus by exploration of this nature can the full outline, both soft tissue and bone, be determined.

ETIOLOGY

Visualization of the topography of the infrabony pocket is an essential for its clinical management, but equally important is an understanding of its causation, for therapy without consideration for correction of the etiologic factors will probably not yield favorable results. Therefore, a detailed clinical examination is obligatory. Tooth anatomy as well as tooth position should be inspected. Teeth without contour associated with heavy wide alveolar processes often show infrabony pockets and vertical resorptive lesions of the bone housing because the disease process is focused to the part directly adjacent to the tooth surface. Mandibular molar teeth are sometimes so affected. The relative uneven levels of adjacent marginal ridges and cemento-enamel junctions with some degree of tilting of the teeth may result in the infrabony type of pocket formation should a gingival disease process be initiated by any local causation (calculus, food impaction, etc.) Thus, the position of the tooth in respect to the alveolar housing and buccal bone, the contact points between teeth and the tilting of teeth must be considered. These and like conditions are local environmental factors which can initiate the infrabony pocket process.

Another important factor in the etiology of the infrabony pocket is the occlusal traumatic lesion. It must be emphasized,

Fig. 15. Radiographs of infrabony pockets with radioopaque material inserted. Note that in this instance in contrast to Figure 14 the bases of the pockets do not coincide with the bottom of the bony defect.

Fig. 16. Radiograph of an infrabony pocket associated with food impaction and an open contact between two molars. Although there are many causes for infrabony pockets, local environmental factors usually contribute to their etiology.

Fig. 17. Radiograph of an infrabony pocket on the mesial aspect of a mandibular third molar. In this instance the second molar had been extracted, allowing the third molar to move mesially. Because of occlusal traumatism and local gingival inflammation due to calculus deposits, and an open contact allowing for food impaction, an infrabony pocket developed.

however, that occlusal traumatism cannot cause pocket formation, since the latter is essentially a soft tissue lesion. There is nevertheless the possibility of the existence of the two factors being present at the same time; first pocket formation caused by any of the local factors (calculus being the most prominent) and second, the occlusal traumatic lesion affecting the attachment apparatus (cementum-periodontal membrane-bone) in the crestal region. With these two factors operating an infrabony pocket will result once the gingival and original transseptal fibers have been destroyed by the continuing inflammatory process.

It is therefore necessary for the operator to understand that infrabony pockets may result from any combination of factors which can allow the base of the pocket to migrate apically alongside the tooth with the bone housing being affected only on one side causing the topographic relationship described previously.

Infrabony pockets also occur in that clinical entity termed periodontosis. Since this disease is found so rarely, the operator should usually base his examination on the above factors. The clinical signs and symptoms of periodontosis are so characteristic that the diagnosis will be soon entertained by the examiner.

THERAPY

Therapy of the infrabony pocket must be directed towards the elimination of the signs and symptoms. For consideration of the elimination of the causation of the infrabony pocket, the following factors should be recognized and corrected, if possible:

1. Tooth anatomy-proximity of roots and width of interdental septum.

2. Relative position of adjacent marginal ridges, cemento-enamel junctions and crest morphology.

3. Tilting of the tooth in group relationships.

4. Tilting of the solitary tooth.

5. Position of the tooth in respect to alveolar housing and basal bone.

6. Contact points and resultant food impaction.

7. The occlusal relationships of the tooth.

8. Presence of calculus.

9. Causation by the disease process periodontosis.

Therefore, correction of the etiologic factors and methods of alleviating symptoms prior to the therapeutic operative procedure

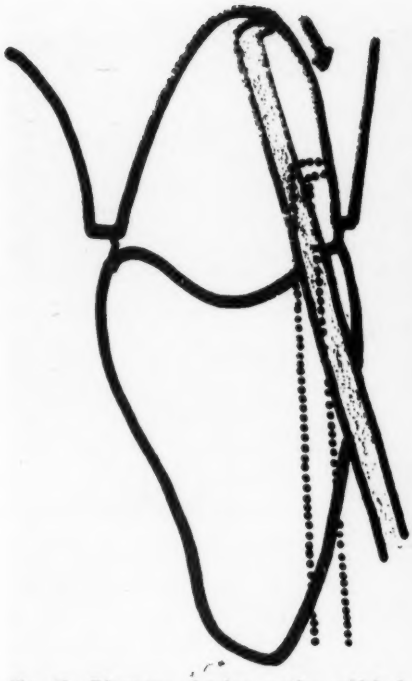

Fig. 19. Diagrams showing motion which is necessary to remove tissue at the base of the pocket. A lingual to buccal or buccal to lingual stroke is very effective, as is seen in this diagram. It is necessary to remove the entire contents of the tooth-bone trough.

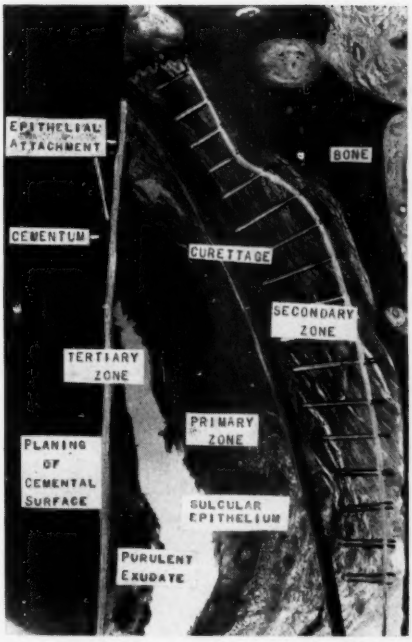

Fig. 20. This is a photomicrograph of an infrabony pocket illustrating the two zones which can be set up arbitrarily in infrabony pockets. The first, sulcular epithelium in the underlying tissue together with the surface of the tooth, and the second, the dense collagen fibers which run over the bone. The importance of the removal of these collagen fibers cannot be overstressed.

may at times be necessary. Tooth anatomy conducive to food impaction, uneven marginal ridges, tilting of teeth and occlusal traumatism should be corrected before the operative procedure to secure a new attachment is attempted. Grinding to correct flat facets, adjustment of the occlusal relationships, levelling of the marginal ridges, placement of restorations to close contact points or to restore physiologic anatomy of the marginal ridges, correction of the occlusal anatomy, by the crowning of a tooth may be necessary. Any attempt to treat an infrabony pocket without regard to the etiologic factor may result in failure.

Should mobility of the tooth be present, temporary splinting should be utilized to stabilize the tooth prior to the operation. Usually orthodontic welded bands are employed although in the anterior part of the dentition, the stainless steel acrylic splint may be used. The mobile tooth should not terminate the temporary splint since this type of splint results in a cantilever effect of the loose tooth. Greater security is obtained by extending the splint from a strongly held tooth to another secure tooth with the mobile tooth in the center. In this way the splint is rigid and the affected tooth cannot be moved by occlusal forces. It must be emphasized that the occlusal relationship must be checked once the splint is placed into position, since any prematurity in occlusal contact on the teeth incorporated in the splint will still cause a movement of the tooth and even may loosen the splint itself. Also important is the fact that occasionally in the cuspid region the splint must be extended around

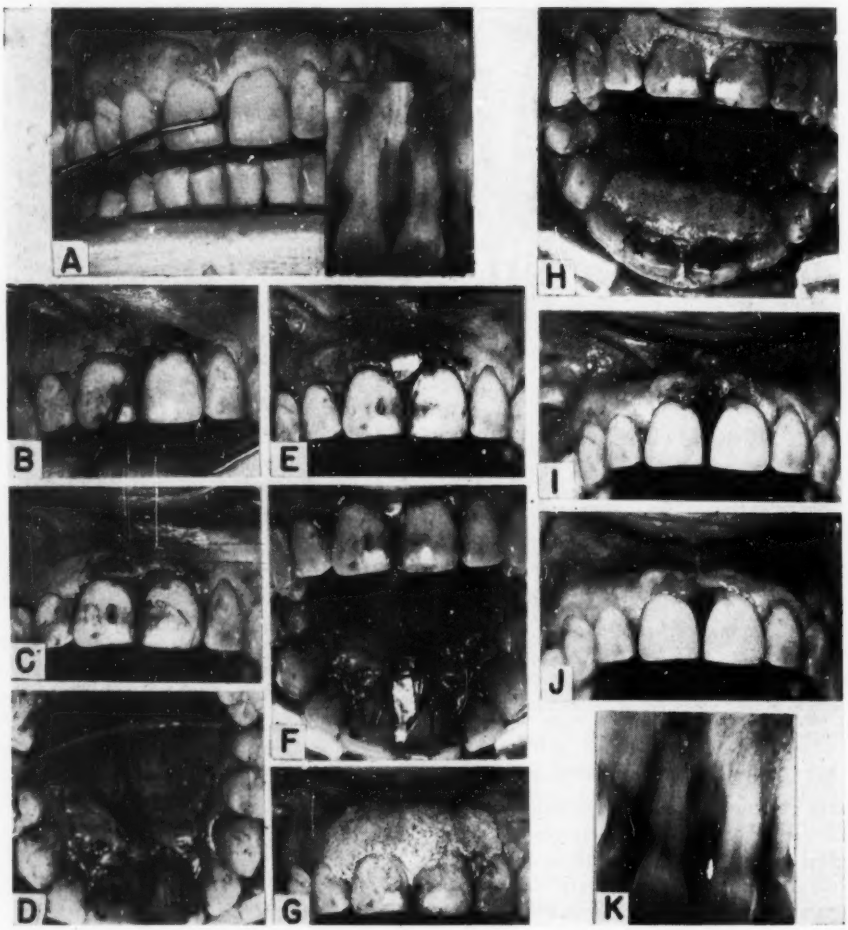

Fig. 18. Photographs of a step-by-step procedure of a three-walled infrabony pocket on the mesial of a maxillary left central incisor. In "A" the pocket is being probed, determining that the buccal and lingual walls were present. The base of the pocket was established at the location of the bony defect. In "B" after the gingival tissue above the bony crest had been excised and the frenum incised, the entire contents of tissue between tooth and bone was removed, results of which can be seen in "C" and "D." Note that a trough-like defect remains. Bleeding occurs and is allowed to continue. The defect is covered with tinfoil in "E" and "F" and in "G" and "H" covered with pack. One week later the pack had been removed (I) and one can note the healing which had taken place. The photograph (J) was taken two weeks after the operation. (K) radiograph taken three months after the time of operation. The gingiva was firm, pink, and adherent. A millimeter probe was inserted into the sulcus and only one millimeter of depth is present. Thus, a healthy sulcus exists. Note the radiographic changes which have occurred in this period of time. The teeth are now in contact, which was not so before operation. Thus, not only was the infrabony pocket eliminated but the involved tooth reverted back into its original position.

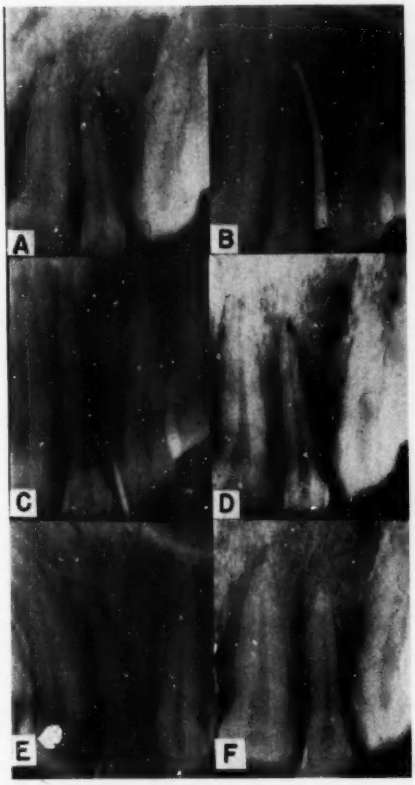

Fig. 21. Radiographs of an infrabony pocket on the distal aspect of a maxillary lateral incisor. The base of the pocket could be located at the apex of the tooth, distal of the tooth but not involving the pulp tissue. The tooth tested vital. After operative procedure "C" a probe could not be inserted. Healing is seen in "D" and "E." Numerous radiographs had been taken throughout the course of ten years, all showing a distinct healing of the bone. A new lamina dura had been established and the tooth was firm and in good position. In "F" the radiograph represents the status of the alveolar supporting structures ten years later.

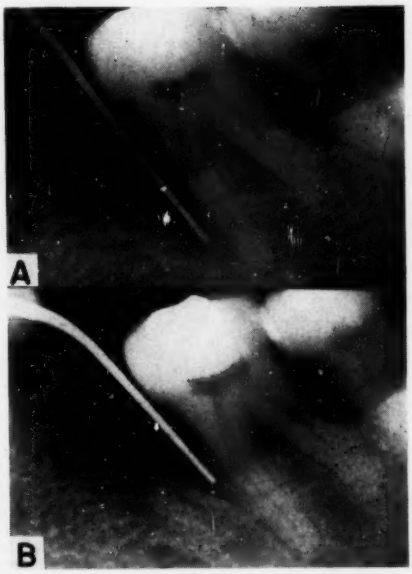

Fig. 22. Before-and-after radiographs showing the healing in the therapy of a three-walled infrabony pocket. One can superimpose one radiograph on another, thus establishing that the measurement of the length of the teeth are exact. Probe shows that there has been several millimeters of regeneration of bone.

the corner of the arch, to the posterior and thereby take advantage of bilateral splinting action.

SELECTION OF THERAPEUTIC PROCEDURE

The objective of treatment is to eliminate the pocket and establish a crevice with as near zero depth as possible.

Two major methods of therapy for the infrabony pocket have been developed. The first consists of curettment of that portion below the bone crest with gingivectomy for the part above the alveolus to enhance the possibility of formation of new cementum, bone and periodontal membrane. The entire tissue between tooth and bone is removed by this debridement. The second method reduces the alveolus to a point coinciding with the base of the pocket; this constitutes an osteoectomy. In order to facilitate accessibility a flap may be reflected to approach the operative site. Of importance in the selection of one of these operative procedures is the evaluation of the amount of remaining alveolus, and the number of lateral walls present. As previously stated, the most favorable type for new attachment is one with the presence of three walls. With two walls a slanting fill in of bone may be obtained whereas when only the proximal wall is present, no additional attachment for the affected tooth may be expected, and osseous surgery is indicated. The length and shape of the

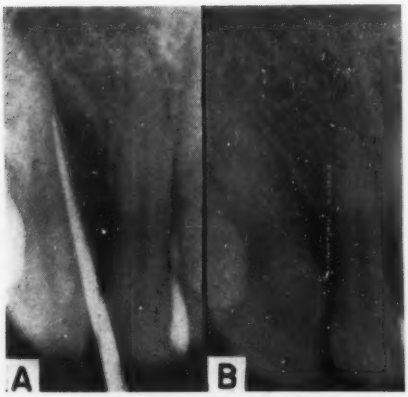

Fig. 23. Before-and-after radiographs showing treatment of an infrabony pocket on the mesial aspect of the maxillary cuspid. Note the establishment of a lamina dura in the after radiograph.

root must be considered in that when a short rooted tooth is to be treated no sacrifice of bony structure may be possible, while should the roots be long and well formed, osteoectomy may be a rational procedure. The housing to the adjacent tooth also plays a role in evaluating the selection of the technical procedure to be used.

As indicated in all periodontal therapeutic techniques for pocket elimination, scaling of the tooth surfaces to remove all deposits should be performed prior to the curettage-gingivectomy operation. Removal of calculus allows for the inflammatory element to be controlled by the repair process, and thus with subsequent therapy, complete repair is enhanced. This holds true for the suprabony portion as well as the infrabony part.

TREATMENT OF THE INFRABONY POCKET WITH THREE OSSEOUS WALLS

— *Curettage-gingivectomy operation for new attachment.* This procedure is carried out under local anesthesia with a minimum or no vasoconstrictor. It should be profound enough to carry out the operation without discomfort to the patient, but still not interfere with the establishment of a blood clot.

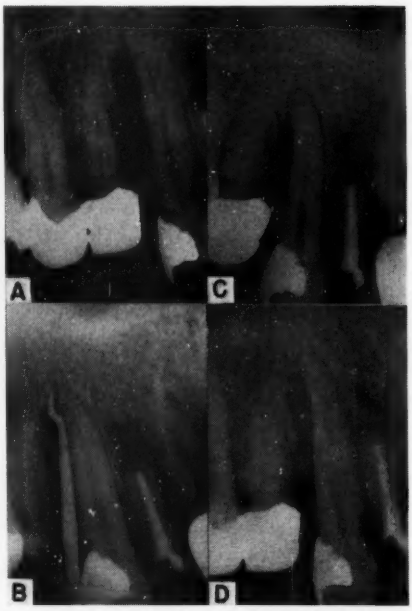

Fig. 24. Radiographs of a three-walled infrabony pocket on the distal of a maxillary cuspid. The base of the pocket could be established almost at the apex of the tooth. Note the filling-in of bone postoperatively.

The gingivectomy phase is performed. The gingiva above the bone crest is excised following the principle of gingivectomy. The resultant gingival margin should be correctly bevelled and the spillways should be accentuated sufficiently. The interdental areas should be pyramidal since the infrabony pocket is included in the treatment of a segment, the adjacent areas will be treated at the same time. Hence the gingivectomy procedure will be performed for the segment; else, the bevelling of the gingivectomy will have to be blended into the adjacent areas.

The next step in this operation is to remove the contents of the pocket by using any curette or scaler. Necessarily the instrument will have to be small, especially in the narrow type of pocket. Often, in addition to a vertical stroke, a horizontal circumferential sweep, short and precise, may be used. Once this has been accomplished, the soft tissue against the osseous wall is

Fig. 25. Photographs and radiographs of a before-and-after case of the therapy of an infrabony pocket around a left central incisor. The tooth tested vital despite the fact that there was an extreme amount of resorption around it. The anterior occlusion was adjusted by grinding. Maxillary and mandibular teeth were shortened so as to create almost an end-to-end occlusion. The procedure was done by raising a flap and removing all the tissue between the tooth and its bony housing. The lateral incisors were moved surgically into position and the flap was readapted. Healing took place rather rapidly.

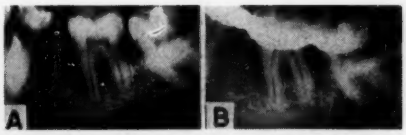

Fig. 26. Before-and-after radiographs of a three-walled infrabony pocket on the mesial aspect of a tilted second mandibular molar. The first molar was extracted many years previously. Orthodontic band splints were made to stabilize the teeth and the operation performed. Note the bone filling in, in the after radiograph (B).

removed with deft, short, cutting strokes. A small sharp curette serves well for this: the circumferential stroke here also works well. The instrument is worked towards the base of the pocket between the tooth and bone, removing all the tissue until the bone wall is felt. Small bits of tissue will be removed easily; at times, a large segment will be excised. The strokes against the bone wall should not be too forceful since if it is curetted further resorption may ensue.

Because of the size of the curettes, even very small ones, the operator should keep the instrument against the tooth side. In this manner the head of the instrument can be placed to the base of the bone-tooth defect. Curettage in this manner will remove the tissue in the portion submerged below the osseous process. It must be stressed that this area must be completely denuded of tissue, else the formation of an attachment apparatus will not occur. It can be seen in Figure 10 that a transseptal fiber group extends to the tooth in the infrabony pocket between tooth and bone. This transseptal fiber group is composed of dense connective tissue fibers running almost parallel to the tooth surface. Hence should only the inner portion of the soft tissue wall be removed leaving these fibers still attached, a blood clot will ensue with healing of the soft tissue wall covered by epithelium. Thus no new attachment takes place. However, if all the tissue is debrided from the area, the blood clot fills the entire area between tooth and bone: after healing

The after photograph may be seen. The gingival architecture was physiologic and the radiograph showed the bone to have filled in completely. The lateral incisor which showed a periapical lesion previously was treated endodontically.

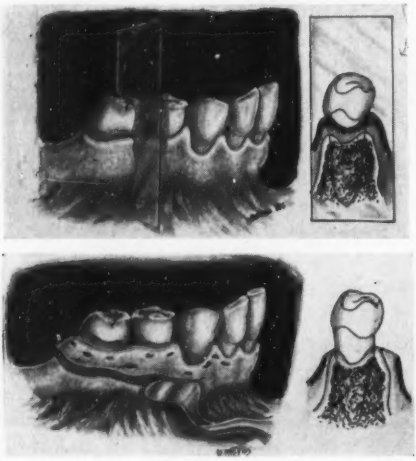

Fig. 27. Drawing of the therapy of a two-walled pocket (crater). In this instance there are buccal and lingual osseous spines with loss of bone interdentally. In the upper drawing one notes the clinical condition, while in the lower one the markings for the gingivectomy may be seen. The gingival tissue is excised. These and following drawings by Dr. Leonard Adams.

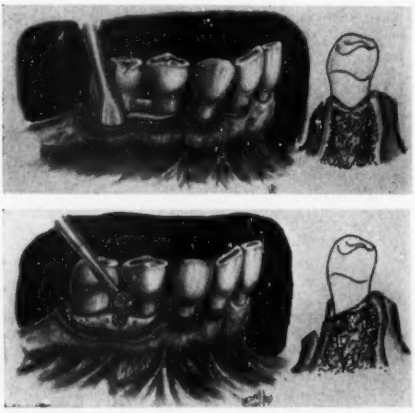

Fig. 28. Continuation of Fig. 27. In the upper drawing the gingival tissue has been excised. The gingival flap is now retracted by the use of a periosteal elevator and alveolar bone is exposed. The bone crest can be removed in many ways: by burs, diamond stones, or hand chisels. In the lower drawing a bur is being used to cut the buccal interdental walls. The bur must be small enough so that it will not engage the tooth interdentally. Also, care must be taken when using burs or diamond stones not to overheat the bone. A water spray is advisable.

a new attachment can form. Thus in the operation one does not consider *only* the removal of the epithelial attachment: in fact this and all the adjacent tissue is removed between tooth and bone up to the region where a normal lamina dura exists. Debridement of the tissue at the base (narrow portion of the infrabony pocket) must be carefully executed since the instrument must be deftly placed and removed with a minimum of trauma to the osseous wall.

Once this phase of the operation is performed, attention is then focused to the tooth. With any suitable instrument, the cleanliness of the tooth should be checked. The operator should be sure that there are no deposits present but also that the surface is smooth. It is possible that gouging defects in the tooth surface will affect healing. Also, caution should be taken that the area above the operative site is not disturbed since the tooth will become sensitive.

When the curettage phase is completed, a trough results, the extent of which depends upon the original topography. This area can be inspected by sponging the area or by using suction to remove the blood. The lateral walls below the bone crest should be checked to ascertain whether any fragmentary tissue has been left. This surface should appear smooth.

Once this procedure has been completed, the trough-like area is allowed to fill with blood and the surface is covered by a small strip of tinfoil or Telfa to prevent any surgical pack from getting into the area. This tinfoil should be securely placed so that it will not be dislodged when the packing is done. The small piece of pack is first inserted interdentally over the foil and then the buccal and lingual packing is placed.

The pack is allowed to remain for one week after which it is repacked: usually the area is protected for about three weeks. After the first packing the area should be filled with tissue and it's not necessary to protect the area with tinfoil or Telfa beneath the pack. The pack is replaced until the tissue surface appears completely epithelialized. At this time the patient is instructed to rinse the area vigorously with hot water.

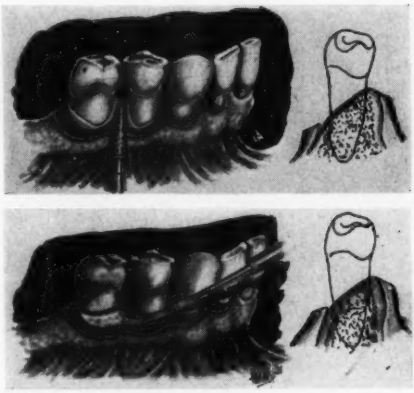

Fig. 29. Continuation of Figs. 27 and 28. Once the buccal alveolar wall has been reduced, hand chisels can then be utilized to trim the bone to desired form. The crestal portion should be bevelled to conform to the desired topography. In this instance the lingual portion is allowed to remain in situ, bevelling the buccal portion to blend into the lingual slope. In the lower photograph a diamond stone is in place showing the cut for the interdental sluiceway.

TREATMENT OF THE INFRABONY POCKET WITH ONE OR TWO OSSEOUS WALLS BY OSTEOECTOMY-OSTEOPLASTY TO ELIMINATE LESION

Successful results by the curettage-gingivectomy operation may not be able to be obtained and in many instances should not be expected (causative agents cannot be eliminated—local environment cannot be changed—three walls not present). As has been stressed, consideration of the bone architecture is important. When *three* walls are present, one may expect almost rou-

Fig. 30. Continuation of Figs. 27-29 showing the covering and pack in position. If zinc oxide is used as a pack, it is wise to use some material adjacent to the bone—either tinfoil or telfa can be utilized for this. To insure the retention of the pack, tinfoil may be used to cover the occlusal surface completely.

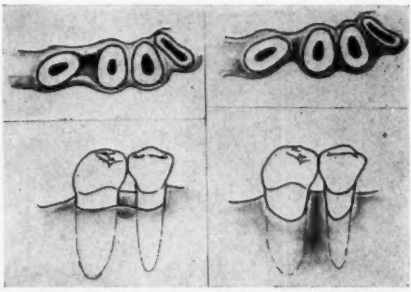

Fig. 31. Before-and-after diagram of the objectives of the operation seen in Figs. 27-30. Note the architecture achieved, comprising the interdental spillway and the gingival position around the teeth. The curvature of the gingival around the tooth, making for physiologic topograph, is essential.

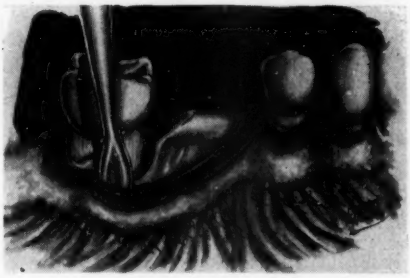

Fig. 32. This is a diagram of the osteoectomy procedure to eliminate a one-walled infrabony pocket. In the upper drawing the gingival tissue has been excised around the premolars at the base of the pocket as it was distal to the molar. However, on the mesial aspect of the molar, the gingival tissue was removed down to a point coincident with the base of the infrabony pocket. This has resulted in the bone, consisting of the mesial wall of the infrabony pocket, being exposed. In the lower drawing this bone tissue is being removed by means of a revolving bone bur. In order to facilitate the removal of this bone, slots are made so that the greater bulk of the osseous structure can be removed by chisel.

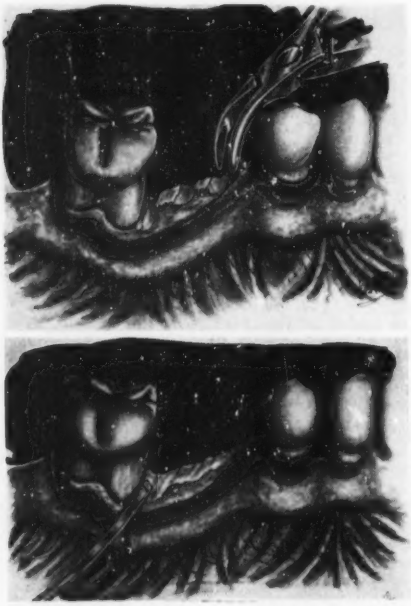

Fig. 33. This is a continuation of the procedure viewed in Fig. 32 (osteoectomy to eliminate a one-walled infrabony pocket). In this instance the bone elevations are being removed by small rongeurs. However, a mallet and chisel or hand chisels also may be utilized to remove the bone. For the final contouring, hand chisels or diamond stones are best employed.

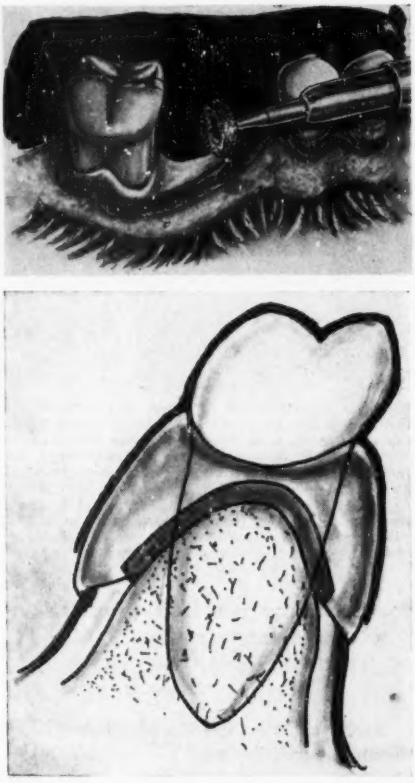

Fig. 34. Continuation of the procedure illustrated in Figs. 32 and 33 (osteoectomy to eliminate a one-walled infrabony pocket). The bone has been recontoured by a revolving diamond stone. Note that the ridge has been contoured so that after healing an acceptable gingival architecture, mesial to the first molar, will ensue. Also the ridge will be able to accept a pontic. In the lower drawing the exposed bone has been covered with telfa and the entire area covered with surgical pack.

tinely that a new attachment will ensue. In a small percentage, failure is encountered; this may result from faulty operative technique, inaccessibility which cannot be overcome or interferences during the healing stage. Often it is wise to reoperate to try to obtain a new attachment. In order to eliminate the infrabony pocket with one or two osseous walls, osteoectomy-osteoplasty procedures are utilized, although at times, the operation for a new attachment may be advisable for pockets with two walls. Not infrequently a "fill" may be obtained.

That procedure which trims the bone crest so that the base of the resultant sulcus will be occlusal to the new bone crest is termed osteoectomy. When recontouring is necessary, the term osteoplasty is used. The decision to eliminate an infrabony pocket by levelling the bone crest must be on a rational basis. The major contraindications are the excessive weakening of the support of an adjacent tooth or the creation of a gingival form not conducive to self-cleansing or difficulty in maintaining cleanliness by oral physiotherapy.

On the other hand, when the pocket is shallow and not too much support is lost, osteoectomy is definitely indicated. Another indication is when a broad infrabony pocket exists on the proximal surface where there is no adjacent tooth present. Occasionally an osteoectomy performed on the buccal crestal bone in the mandibular molar

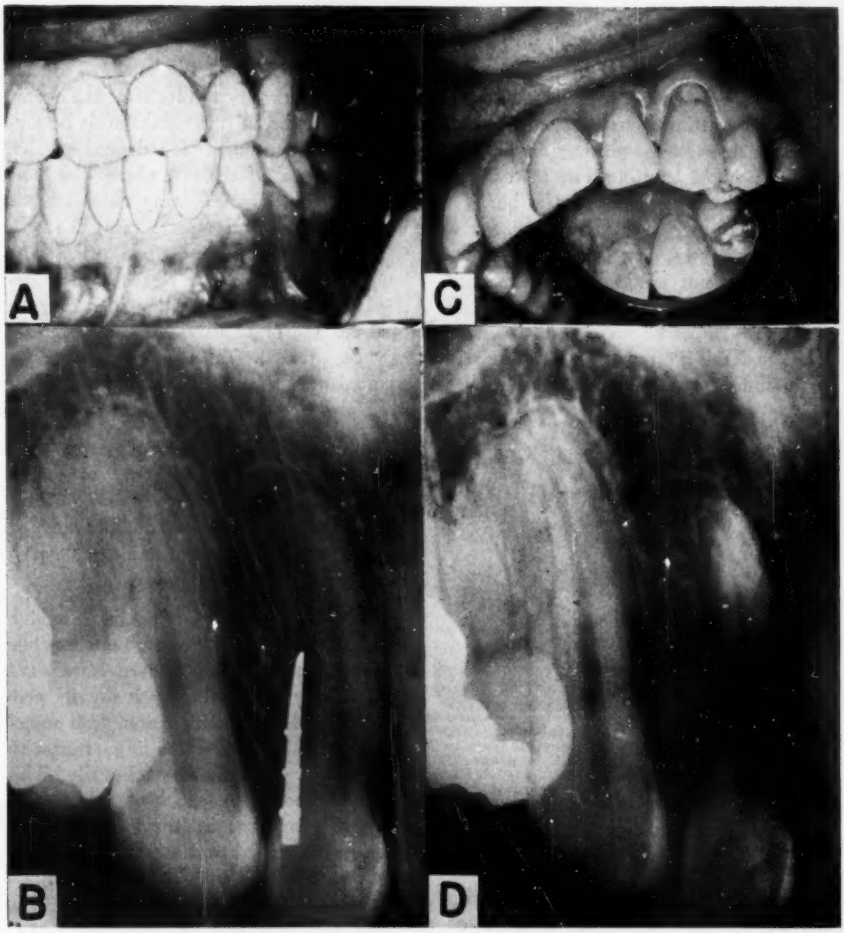

Fig. 35. Photographs and radiographs of the treatment of a one-walled infrabony pocket distal to the maxillary left lateral incisor may be seen. The prognosis for the one-walled pocket in this instance was negative and, hence, an osteoectomy was performed. The gingival tissue was excised following which the crest of the bone on the cuspid was removed, levelling it off between the two teeth. The gingival tissue was recontoured so that no food retention would take place. In the after photograph (C) the gingival contour may be seen whereas in "D" the final radiograph is shown.

region where an infrabony pocket extends into the interradicular area allows for the elimination of the pocket with resultant acceptable architecture. Contouring the bifurcation area so that food accumulation will not occur aids cleansing in that area. Many such examples can be cited; when indicated this procedure is a very valuable asset in the armamentarium for pocket elimination.

There are two major procedures for osteoectomy-osteoplasty. The first comprises the raising of a flap, full or modified, and subsequent trimming of the bone crest. After reflecting the flap, the base of the pocket is measured and marked off on the outer bone wall. The bone may be chiselled away with small enamel chisels or may be cut away by bur in a dental engine. One advantage for the use of a chisel is that it

can be easily maneuvered interproximally and thereby reduce the possibility of gouging tooth structure which may occur with burs or stones. Another method is to make small holes, outline the contour of the bone for removal, and then uniting the holes by chisel. The bone contour can then be accomplished by either the chisel or bur. The operator, however, must be warned that when a bur is used, care must be taken that the tooth is not engaged less defects be made. Bone files, necessarily small, are also useful: they must be sharp and maneuverable. The debris is removed by flushing with warm saline water. Once the base is reached,

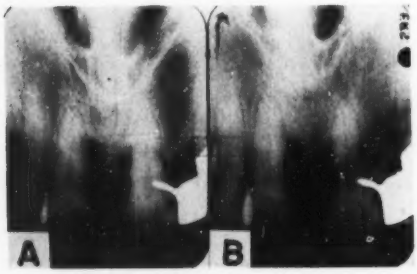

Fig. 36. Radiographs of two infrabony pockets (A) mesial to the maxillary central incisors. These were one-walled pockets and the prognosis for regeneration was negative. An osteoectomy was performed removing the interdental spur. The gingival tissue was then recontoured creating a saddle area in this region. The gingivae healed, becoming firm, pink, and well-attached.

the crest should be rounded and smoothened by diamond stones so that no deformity will occur once the gingival flap is replaced. Mention must be made that the inner wall of the gingival flap must be debrided before replacement. The flap should be carefully replaced and tautly and securely sutured into place: it is often necessary to trim the gingiva in order not to have too much tissue in the interdental areas and not to allow the flaps to be loosely readapted. A mattress suture is often helpful.

The other method is to mark off the base of the pocket on the outer surface by the use of a modified Crane-Kaplan pocket marker; a probe can also be used to demarcate the outline of the pocket and then to record the markings on the outer gingivae. A gingivectomy should be outlined so that no severe deformity results after the operation. Thus, the adjacent areas must be surveyed and contoured for desired results. After removal of the soft tissues the bone crest is denuded by reflecting the soft tissue with a periosteal elevator placing a small, sharp chisel at an acute angle against the bone crest, the bone can be trimmed to desired proportions. A small mallet may be used for the blow although hand pressure can be utilized. Telfa is then carefully adapted over the bone and then a surgical pack placed.

Each method has its advantages and disadvantages. The gingivectomy procedure offers the opportunity for obtaining a better gingival topography and also any of the fold extension operations can be performed simultaneously.

—The two wall infrabony pocket in which the buccal and lingual walls remain while the proximal wall is destroyed is referred to as a crater. Osteoectomy is usually performed to eliminate this lesion. It is sometimes desirable to allow the lingual crest to remain and ramp the crest bucally. This does not produce too much of a deformity and still allows the patient to cleanse and massage the interdental area where the tip of the papilla has been placed in the lingual part of the interproximal area.

—The infrabony pocket with one osseous wall is also best treated by the osteoectomy-osteoplasty procedure. The gingivectomy is performed and then the tissue is reflected revealing the infrabony pocket. Usually the one remaining wall is a proximal septum and this is eliminated by chisels or burs. The osseous tissue is smoothed with diamond stones before placing the telfa and pack.

TREATMENT OF THE COMBINATION TYPES OF INFRABONY POCKETS

The clinician will encounter infrabony pockets where the number of osseous walls

may vary in different parts of the same pocket. Most frequently 3 walls will be observed at the apical part of the pocket with one and/or two osseous walls at the coronal part of the pocket. In these situations the one or two wall part of the pocket is eliminated by osseous surgery while the three wall part is prepared for new attachment by the curettage-gingivectomy procedure. Thus combinations of procedures are selected depending on the morphology of these combination types of infrabony pockets.

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MRS. LORRIE HILL HONORED

All members of the American Academy of Periodontology are familiar with the efficient manner in which Mrs. Lorrie Hill assists Dr. Clarke E. Chamberlain in his many activities as Secretary of the American Academy of Periodontology. It was not surprising therefore to learn that recently the National Secretaries Association recognized Mrs. Hill's ability, and awarded her their coveted Certified Professional Secretary Award. Congratulations, Mrs. Hill!

The Effects of Stress on the Periodontium of the Syrian Hamster

by PETER F. FEDI, JR.,* D.D.S., M.SC., *Columbus, Ohio*

NUMEROUS local and systemic factors which may affect the periodontium have been investigated. Local factors, such as calculus, faulty restorations, and trauma, have been shown to produce definite changes in the periodontium.[10, 16, 24, 25, 35] Diabetes, hormonal disturbances, and nutritional deficiencies have been studied at some length[3, 4, 6, 7, 8, 9, 10, 11, 18, 20, 23, 24, 25, 26, 34, 35] but the investigators limited their studies to specific factors which might affect the periodontium.

This paper is based on a thesis submitted in partial fulfillment of the requirements for the Master of Science Degree at Ohio State University, June 1956.

Few investigators have directed their studies toward non-specific factors as possible causes of periodontal disease. The relationship between non-specific factors and the periodontium should be clarified through investigation so that the periodontist may utilize the knowledge so obtained for prevention and therapy.

Stress has been associated with non-specific somatic changes. The sum of the systemic reactions of the body which result from long and continuous stress has been termed the "general adaptation syndrome."[27, 28, 29, 30, 31, 32] Selye described the general adaptation syndrome (GAS) as the basis for a concept associated with the pathogenesis of many diseases that previously were designated as being of unknown etiology, and to explain the common denominator of being "sick." The purpose of this study was to determine the effect of stress on the periodontium of the Syrian hamster as a pilot study for future work on stress in its relationship to periodontal disease. It is the intent of the author, that this paper be considered a report of a pilot study. It will be noted that there are many factors which would affect the validity of the results and which must be made an integral part of any future study in this field (i.e., the use of litter mate animals, autopsy studies of vital organs, etc.).

DEFINITIONS

For the purpose of this paper the following definitions have been used:

Specific factors are most agents which affect only a very limited number of "targets" with great selectivity and which produce specific changes, i.e., diabetes mellitus.

Non-specific factors are most agents which affect many "targets" and are devoid of ability to act selectively upon any one. They produce non-specific changes, i.e., extreme cold.

General Adaptation Syndrome (GAS) is a triphasic, non-specific adaptive reaction comprised of the alarm reaction, the stage of resistance and the stage of exhaustion.

Stress is the sum of all non-specific biological phenomena (including damage and defense). It may be local or systemic. The term stress is used as the condition while a *stressor* is the agent which causes the condition of stress.

Target is the tissue, organ or region primarily affected by the stressor.

Antiphlogistic agent is one which counteracts inflammation, i.e., glucocorticoids.

Prophlogistic agent is one which enhances inflammation, i.e., mineralocorticoids.

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The opinions or assertions contained in this article are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department of the Naval Service at large.

Selye's concept arose from observations that many diseases have a number of common features. While diseases themselves may be quite specific, the common features usually are not so well defined. Selye elucidated the concept of stress/with unification as its leading element. The common, non-specific features of disease are closely integrated and represent part of a single syndrome. Non-specific stressors and specific diseases associated with infection, trauma, heat and other specific causes have a common feature which places the body in a state of general systemic stress.

Stress affects the body in many ways but the triphasic reaction is adrenocortical enlargement, thymicolymphatic involution and gastro-intestinal ulcers. All of the organs of the body except the adrenal cortex, show some involution or degeneration. The single response to stress (the General Adaptation Syndrome) can be divided into three distinct phases:

The Alarm Reaction—The period between the beginning of the stress and the time when the body begins to adapt to these new circumstances.

The stage of Resistance—The period following the Alarm Reaction, during which the body continues to increase its adaptive powers until it has done as much as it can and continues to resist in an optimal manner.

The stage of Exhaustion—The period at which the body's resistance is finally overcome by the loss of the required adaptation due to continued stress.

The stressor (Figure 1) stresses the body by setting off a chain of reactions in which the adrenal cortex is stimulated via the adrenocorticotropic hormone (ACTH). The ACTH is produced by the anterior portion of the pituitary gland along with somatotropic hormone (STH). The adrenal cortex, stimulated by ACTH, produces both prophlogistic mineralocorticoids, such as desoxycorticosterone, and anti-phlogistic gluco-corticoids. The STH acts in a manner similar to mineralocorticoids and it is believed that the response of the tissues to STH is sensitization of the peripheral tissues to the mineralocorticoids.

The general adaptation syndrome theory and the concept of non-specific stresses proposed by Selye[31] gave support to the belief that these non-specific diseases of adaptation may play a role in osteoporosis of the alveolar process and in degeneration of the periodontal membrane.

Ratcliff,[21] investigated the relationship of the periodontal tissues to non-specific adverse influences on the body and attempted to determine what changes might occur in the periodontium in the more advanced stages of the GAS. He produced stress by frustration, fatigue and hunger. The animals were sacrificed after stressing for 68 hours. Nine of the 10 experimental animals were in the stage of resistance and the remaining rat was in the stage of exhaustion. No histological differences in the periodontal tissues were found but the changes were more pronounced during the third stage. Sloughing of the keratinized layers of the epithelium occurred in the gingival tissues. A split of the papilla occurred at the junction of the oral and enamel epithelial cells and degeneration of the connective tissue of the periodontal membrane was evident. Osteoblasts and cementoblasts were reduced in number.

Shklar and Glickman[33] reported no changes in the periodontal structures of rats stressed by subcutaneous injections of 4% formaldehyde. They injected 5 cc.'s four times in 48 hours. These investigators also withheld food for the 48-hour period of the experiment and they reported that the animals were in a typical alarm reaction of the GAS.

Zander and Muhlemann[36] applied mechanical forces of an orthodontic nature and subjected monkeys to systemic stressors. They showed necrosis, degeneration, circulatory disturbances, osteoclasia, and formation of new bone. The changes were most pronounced in the marginal and apical

pressure zones and marginal tension zones of the periodontal membrane. They also revealed that there appeared to be a "normal" osteoclastic activity as well as physiologic bone apposition although in many places there was found perivascular edema combined with discrete round cell extravasation. These authors also stated that neither the systemically stressed nor the non-stressed monkeys showed symptoms of periodontal disease comparable to human conditions.

Glickman, Stone and Chawla[14] noted osteoporosis of the alveolar bone, degeneration and reduction in the number of the collagen fibers of the periodontal membrane of animals subjected to repeated injection of cortisone. Reduced osteoblastic activity in the skeletal system in areas other than the alveolar bone has been reported following injections with ACTH and cortisone.[2]

THE EXPERIMENTAL METHOD

Twenty-four young, weaned, female, Syrian hamsters were used. Their diet consisted of Purina dog chow in pellet form, with a constant water supply. The average weight upon arrival was 60 grams.

The animals were placed in clear plastic cages, 4 animals to a cage, with a bed of cedar chips and a perforated metal top. To be assured that all animals would start the experiment as free of stress as possible, they were kept as pets for 2 weeks after arrival. Daily handling and play was employed with hand feeding of celery, lettuce leaves and carrots along with their daily ration of Purina chow and water. At the end of this 2-week period all 24 animals were high spirited and friendly pets. They could be handled with ease and seemed to look forward to their daily handling. At this time, 6 animals, picked at random, were placed in 2 cages, 3 to a cage. These animals were the control animals and were treated throughout the experiment as pets.

The remaining 18 were used as experimental animals. It was thought desirable to keep these animals under a stressed condition for 6 weeks. This was done in accordance with work carried out by Selye[21] and Pennel.[19] All experimental animals underwent 3 types or phases of stressor application. The first two-week period consisted of daily exposure to cold, the second two-week period consisted of daily exposure to a water bath and violent exercise, and the third two-week period consisted of daily subcutaneous injections of 10 per cent formalin.

The thermal treatment was carried out by placing 4 animals in a plastic cage that was partitioned so that the animals would neither see each other nor

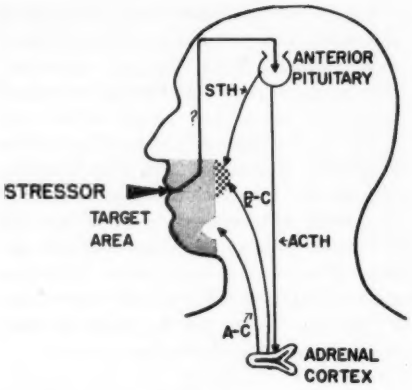

Figure 1

gain warmth by body contact. The cage was fitted with a perforated metal top and placed in a deep freezer at zero degree Fahrenheit in an inverted position, so that the metal top became the floor of the cage and rested upon the cold metal racks of the freezer. They were kept at this temperature and position for 30 minutes a day. While the animals were in the freezer they tried desperately to keep their unprotected feet warm by licking them, and also tried to maintain a position above the metal floor of the cage by grasping the uppermost edge of the partition. They could maintain this position for only a few minutes at a time. Upon removal the animals displayed signs of complete fatigue. It was found that the animals displayed a typical reflex phenomenon after the second or third day. They would start licking their feet immediately upon being placed in the special freezer cage. By the end of the two-week period of stress by exposure to cold, 5 animals had died. Of the remaining 13 animals, 4 were extremely belligerent and could not be handled in the routine manner. Instead of carrying on a normal type of play, these animals would try to kill their cage mates and had to be separated from each other.

Stress induction by exposure to a water bath and violent exercise consisted of placing the animals in a large laboratory sink filled with water that averaged 48 degrees Fahrenheit. For the first 2 days the animals were placed in warm water, but most of them seemed to enjoy the exercise, although they did not care for the water bath, so it was decided that cold water might increase the stress effect. The majority of the animals could barely stay afloat at the end of 5 minutes in 48 degrees (Fahrenheit) water. Upon removal from the cold swim, the animals were so exhausted they could be handled easily. They also were so cold they became stiff and if placed on their backs in their cages, they would remain in this position for approximately 5 minutes. When they

had recovered from the cold, they would attempt to clean their fur and immediately lie down and sleep. By the time of termination of this phase of the experiment the animals would growl and attempt to bite the hand of anyone approaching their cages. During the course of this phase of experimentation, many animals had to be given some form of artificial respiration after near death by drowning. None of the experimental animals died during this phase of stressor application.

The third phase of stressor application began by injecting 4 tenths of a cc. of a 10 per cent formalin solution subcutaneously in the loose skin on the back of the animals. This site of application could not be reached by the animals and therefore served two purposes. They could not aggravate the site of injection by scratching, and since they could not reach the area of injury, they were subjected to the mental torment of not being able to effectively touch the injury and were forced to resort to such other methods as they could devise. A few seconds after injection, the animals would make continuing gyrations for several minutes in an attempt to scratch the area of injury. They would resort to such things as diving in and out of their cedar chip beds, jumping at the top of their cages, somersaulting in mid-air and landing on their backs, biting and scratching each other until blood appeared, at which time the animals would beat a hasty retreat.

It became increasingly difficult to handle and inject these ferocious animals, for by this time they had become extremely pugnacious. A method was devised, whereby safe injections could be made exactly where they were needed. The animals were scooped out of their cage in a large tea strainer and placed in cold water for three minutes. Then the animals became stiff and almost immovable. It was now possible to inject the formalin and place the animals back in their respective cages with ease. Most of the animals would go into a convulsive-like state, within minutes after being replaced in their cages and one animal would repeatedly salivate profusely during the convulsions. During this phase of stressor application, 6 experimental animals died. One control animal died during this period of time and the cause of death was not determined upon autopsy.

Upon termination of the experiment the 7 remaining experimental animals and the 5 remaining control animals were sacrificed. The maxillae and mandibles were removed in toto and prepared for sectioning. Bucco lingual sections in the mandibular molar regions were prepared at 10 microns and stained with hematoxylin and eosin.

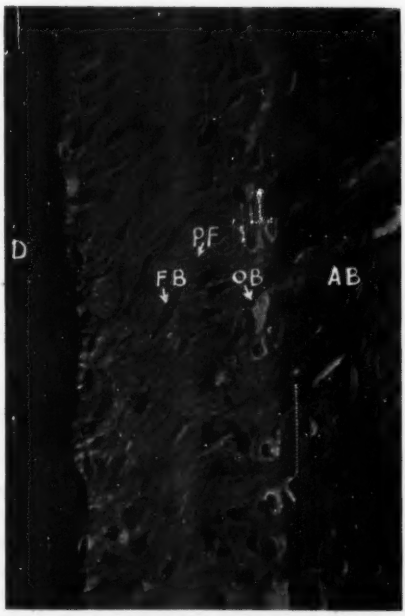

Fig. 2. Experimental Section of Periodontal Membrane. D—Dentin; AB—Alveolar Bone; OB—Odontoblast; FB—Fibroblast; PF—Principal Fibers of Periodontal ligament.

EXPERIMENTAL RESULTS

Gross observations of the teeth and jaws of the hamsters after decapitation, showed no significant differences between the control and experimental animals. The microscopic sections were studied and at first there seemed to be no striking differences between comparable sections of control or experimental animals.

Upon further examination, it was noted that the principal fibers of the periodontal membrane seemed to be coarser in the experimental sections than in the control sections (Figures 2 and 3). These principal fibers of the periodontal membrane in experimental animals seemed to have less regularity of arrangement. They seemed to disperse in all directions in the experimental sections while in the control sections the fibers had a uniform regularity in direction and in relationship of one group of fibers to the other.

In some areas of the experimental sections, the fibroblasts appeared to be more mature and irregular in shape. Spindle shaped fibroblasts with comparatively larger nuclei and well defined nuclear gran-

ules predominated in the control sections. The experimental sections showed what appeared to be many round fibroblasts with smaller nuclei (often pyknotic) and less nuclear granular material interspersed with but a few of the typical spindle shaped fibroblasts.

In some areas of the experimental sections, the new bone formation appeared to be irregular (Figures 4 and 5). The new bone also appeared to pick up the hematoxylin and eosin stain in an irregular manner. Some areas of bone appeared extremely dark while others were rather pale and this mixture presented itself in a conglomeration of varying hues of tissue arranged in a mosaic-like pattern. The osteoblasts appeared to be somewhat rounded, to have more pyknotic nuclei, and to be less regular in arrangement than in the control sections (Figures 2 and 3).

On both the control and experimental sections the distal alveolar walls of the molar teeth showed generalized resorption of bone with numerous Howship's lacunae, many of which had osteoclasts adjacent to them. On the mesial alveolar walls, in both sections, there appeared to be a generalized apposition of bone (Figure 6).

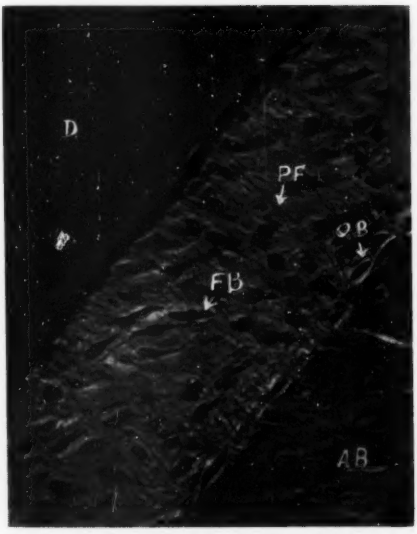

Fig. 3. Control Section of Periodontal Membrane. D—Dentin; AB—Alveolar Bone; OB—Odontoblast; FB—Fibroblast; PF—Principal Fiber of Periodontal ligament.

DISCUSSION

Osteoporosis is a common finding associated with the alarm reaction following trauma, protein deficiency, or other types of stress.[10] The cause may be catabolism of existing osteoid or disturbance in osteoid formation. Albright and Reifenstein [22] stated that the major causes of bone dissolution during the general adaptation syndrone is the shift of pituitary secretion, and the change of hormone equilibrium with increased production of glucocorticoids. This increase in glucocorticoids was assumed to be due to protein catabolism and osteoporosis. The osteoporosis of Cushings syndrome is apparently the result of such hormonal disbalance and is quite similar to the osteoporosis of the general adaptation syndrome. Glickman suggested[10] that the significance of the hormonal pattern producing osteoporosis is qualified by the fact that comparable, but less severe osteoporosis occurs in disease states in which bone has been immobilized, and that osteoporosis has been shown to occur in the absence of both the pituitary and the adrenals. He theorized that probably the hormonal changes in the alarm reaction merely facilitate certain preexisting metabolic processes.

Glickman has discussed the general concept of bone change under the title of "bone factor concept."[10,12] He has suggested that there, the relationship between the alveolar bone and the remainder of the skeletal system is manifested in the clinical pattern of chronic destructive periodontal disease. He has demonstrated[8,9,10,11,12,13,15] the manner in which different systemic influences alter the response of the alveolar bone to local irritation and the manner in which the different systemic influences result in the destruction of the periodontal tissue in the absence of local irritants. Glickman has stated the belief

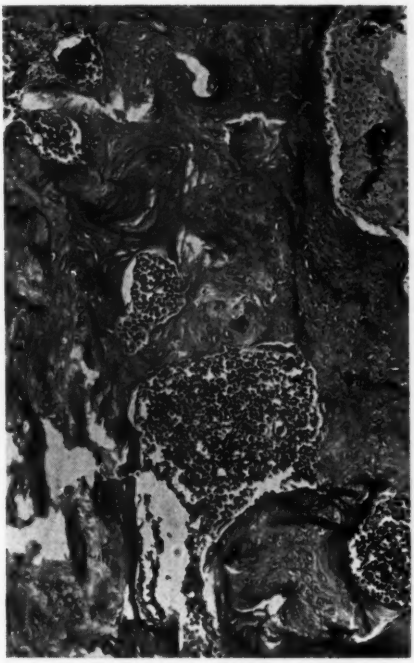

Fig. 4. Experimental Section of Mandibular Bone. "Heteromorphic" Bone pattern.

Fig. 5. Control Section of Mandibular Bone. "Normal" Bone pattern.

that there are two effects of stress upon growth and osteoporosis:[10] Somatic growth is inhibited during exposure to stress particularly in the alarm reaction stage and in the stage of exhaustion of the GAS. This is believed to be due to a shift in the pituitary activity whereby ACTH is produced at the expense of STH production.

In our study there did not appear to be any significant reduction in the number of osteoblasts nor any increase in the number of osteoclasts. This is contrary to the results of Ratcliff[21] and of Pennel.[19] The control and experimental sections showed approximately equal osteoblastic activity with apparent similarity in the number of osteoblasts in comparable sections. The osteoblasts in the experimental sections did show more variations than those in the control sections. Osteoblasts, and bone resorption, were found in approximately the same amounts in the control and experimental sections. It is possible that these findings were contrary to those of Ratcliff and Pennel's because they used rats as their experimental animals and the Syrian hamster was used in this experiment. Syrian hamster molar teeth more nearly resemble the human molar in shape, and the rat is known to be acclimated to its environment quite readily.[19] While little is known concerning the ability of hamsters to withstand stress it appears from this study that they are readily subject to non-specific stressors.

Changes that did occur in the bone, under the influence of stress other than cytological changes noted, are best described as formation of heteromorphic bone. The bone pattern in the stressed animals was characterized by more irregular bone formation than in the control animals.

The changes of the stressed animals were significant in the periodontal membranes.

The principal fibers of the periodontal membrane appeared to be coarser than those in the control animals. There also was less regularity in arrangement of principal fibers of the periodontal membrane. The fibroblasts appeared to be more mature. The nuclei of many of the fibroblasts were smaller and in some cases they were actually pyknotic, the nuclear granules of the fibroblasts being more prominent than in the fibroblasts of the control animals. Doughtery and Higginbotham,[30] following hydrocortisone administration to non-inflamed loose connective tissues found that the fibroblasts pulled in their processes and tended to round up and resemble epithelial cells. No such changes were found for the clasmatocytes, lymphocytes, mast cells, or eosinphils. They assumed that the fibroblasts are the most active cells in sequestering locally administered anti-inflammatory steroid hormone. Doughtery and Higginbotham have shown that the antiphlogistic corticoids inhibit the inflammatory responses to certain stimuli. This observation of changes in the fibroblasts of the periodontal membrane of the stressed animals is in general accord with the suggestions of Doughtery and Higginbotham. However, there was no evidence of change to epithelioid type cells in the periodontal membrane of these animals.

While it was not a portion of the study of stress, the observation of apposition of alveolar bone on the mesial alveolar surface and the resorption of bone on the distal surfaces of the molar teeth in these animals is interesting in that it is the opposite of that which occurs in the human dentition.[17] This finding suggests that in Syrian hamsters the physiological drift may be in a distal direction, but further study of this is indicated before final conclusions should be made.

Fig. 6. Control Section of Interproximal Alveolar Bone. AB—Alveolar Bone; D—Dentin; PDM —Periodontal ligament.

SUMMARY AND CONCLUSIONS

The purpose of this study was to determine the effects of stress upon the alveolar bone of the Syrian hamster. Twenty-four Syrian hamsters were used. Six were used as controls and 18 animals were subjected to non-specific adverse influences. The Syrian hamsters that were subjected to experimental stressors, exhibited a non-specific stress reaction. Microscopically, the principal fibers of the periodontal membrane appeared to be coarser, there was less regularity in arrangement of the principal fibers of the periodontal membrane, in some regions, the fibroblasts appeared to be more mature, new bone formation in some areas seemed to be irregular and the bone could be described as heteromorphic and the osteoblasts appear to be somewhat rounded, to have more pyknotic nuclei, and to be less regular in arrangement.

These findings indicate that the alveolar bone and periodontal membrane of the Syrian hamsters are affected by non-specific stress.

It was also observed that, contrary to findings of other investigators that there

was no apparent decrease in the number of osteoblasts, no apparent increase in the number of osteoclasts, no significant decrease in the number of collagen fibers and no apparent decrease in the number of cementoblasts. It may be possible that the reason for this is that the Syrian hamster was used for this experiment, while the other investigators used the rat.

It was interesting to note that the distal alveolar bone was in a state of resorption and the mesial alveolar bone was in a state of apposition. This tends to suggest the possibility that hamster molars have a distal drift.

The author wishes to express his appreciation to Drs. John R. Wilson, Hamilton B. G. Robinson and William Lefkowitz for their guidance and cooperation in conduct of the research project on which this paper is based.

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Effect of the Administration of Steroid Hormones on the Gingival Tissues*

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SINCE the introduction of the steroid compounds a few years ago, there has been much speculation as to their possible effects on periodontal disease. This speculation can best be exemplified by the nature of the various reports appearing in the literature.[1-20] Much of the literature reports the effect of steroid administration on a selected few cases.[7-18, 20-23] Other reports list the effects of steroid administration on animals and attempt to transpose the results to human beings.[19, 24-28] Selye,[1] using the granuloma pouch technique, found that these drugs inhibited the development of inflammatory processes. Steroids have been used to treat systemic diseases because fibroblastic proliferation is thought to be inhibited as well as osteogenesis and granulation tissue formation.[2]

Because of the paucity of the literature on this subject, and the possible effect of these drugs on periodontal disease, it was deemed desirable to study a large series of patients. For this report an examination was made of the gingival tissues of a large number of patients receiving significant doses of the steroid drugs for the treatment of systemic diseases. The gingival tissues of these patients were then compared with the gingival tissues of similar patients not receiving these drugs, in order to determine whether there was a significant effect of the steroid compounds on the gingival tissue.

*A Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of Master of Science in Periodontics.

REVIEW OF THE LITERATURE

Cortisone and ACTH produce similar effects but in different ways. One substitutes for the gland and the other stimulates it.[5] One report cautions that care must be exercised in the use of steroid therapy because by suppressing the signs and symptoms of inflammation, the drug may mask or intensify the progress of infection.[4] Shroff says that the evidence concerning effects of steroid hormones in the cause of, or as modifying agents in the course of, periodontal diseases is at present indefinite.[6]

Fox and Stahl[9] studied the effect on the epithelium, basement membrane and connective tissue of small amounts of hydrocortisone injected into normal human oral mucosa. They used .05 cc. of a 2.5% solution. They also applied 2.5% hydrocortisone acetate ointment to the gingival papillae once a day for three days. They used untreated papillae as controls and examined the tissues at intervals of ten minutes to 216 hours following treatment. They found no distinct histologic local change except "a transient reduction in cellularity of the connective tissue of the lamina propria, and an equally transient irregularity of the basement membrane."

In 79 endodontic cases, Wolfsohn[7] found a reduction and elimination of severe inflammatory reactions in the periodontal membrane following treatment, as long as the chambers were sterilized first. They used .5 cc. of hydrocortisone aqueous saline suspension. The similar use of hydrocortisone at the conclusion of immediate pulpectomy procedures resulted in diminution and absence of secondary trauma of the periodontal membrane. This drug, however, when used in the presence of infected pulps and periapical tissues resulted in exacerba-

tion of the disease process.

Strean and Horton[8] reported that in ten successive cases of root canal therapy, there were no symptoms of pain in the periapical areas where the canals had at one point been saturated with a hydrocortisone saline suspension, which was pumped through the apex.

Strean and Horton[9] also reported that in five cases of temporomandibular joint arthritis, symptomatic relief was obtained by an injection of .5 cc. of an hydrocortisone acetate solution in the joint itself. Dreizen et al,[10] claimed that three cases of acute arthritis of the temporomandibular joint responded by treatment with ACTH or cortisone. Horton,[11] too, claimed relief of three such patients with these drugs. There was a marked reduction of inflammation in three other cases of glossitis, denture sore mouth, and an immediate denture case, respectively, according to Horton, after hydrocortisone ointments of 1% to 2.5% were applied for a few days. However, Doolen[12] suggests that in one case there was a marked reduction of healing after extractions due to the systemic administration of 100 mg. of cortisone daily for the treatment of arthritis.

Bergmen[13] reported that of 17 patients treated with hydrocortisone for aphthous ulcers on the lips or intra-orally, 15 responded by the elimination of pain within one day, and prompt healing of the ulcer. Dreizen, et al,[14] claimed that in four patients with stomatitis and dermatitis due to allergy, the oral and facial lesions healed by the use of ACTH or cortisone. In two patients with psoriasis there was a decrease in the severity of the facial lesions with the use of these drugs. In the treatment of disseminated lupus, the extensive oral and facial lesions decreased noticeably. They also reported that the severity of oral lesions in acute lymphatic leukemia decreased following injections of ACTH.

Strean and Horton[15] reported that five cases of desquamative gingivitis showed demonstrable improvement after a few weeks treatment with hydrocortisone ointment. Strean[16] reported on ten cases of desquamative gingivitis which responded to either topical application of hydrocortisone ointment or by local injection of 6.25 mg. of cortisone in .25 cc. of saline. Dreizen, et al,[17] also reported the remission of gingivitis in two patients with hormonal desquamative gingivitis. They said that one 200 mg. cortisone injection intra-muscularly wasn't curative, but exerted detectable influence on pathologic physiology.

Bain[18] reported elimination of inflammatory symptoms and return to normal in five cases of pregnancy gingivitis.

Staple[19] suggests that the gingival hyperplasia seen in patients given sodium dilantin may be the result of adrenocortical dysfunction, as a result of some work done on ferrets. Strean and Horton[20] reported that the hyperplastic tissue of dilantin gingivitis did not return after surgery, as long as the hydrocortisone ointment was applied twice a day for a period of three months. They theorized that this was due to its effect on the inhibition of fibroblastic proliferation. However, Sackler, et al,[21] reported on the use of a 2.5% hydrocortisone acetate ointment on a case of hyperplastic gingivitis due to dilantin, and found it did not decrease the gingival hyperplasia, nor did it inhibit the inflammatory reaction.

Strean and Horton[22] reported an undisclosed number of patients with gingival inflammation due to irritation. They said that inflammation subsided several days after rubbing a 2.5% hydrocortisone acetate ointment on the gingivae twice a day.

Iusem[23] et al said that cortisone has been found to be useful in (a) diminishing capillary permeability, polymorphonuclear diapedesis, and inhibition of collagen; (b) reducing the number of plasma cells found in granulation tissue and lymphocytic proliferative tissues. They used gingival injections of cortisone and reported improvement in hemorrhagic hyperplastic gingivitis and reduction of chronic granulation tissue found in periodontal pockets and abscesses. They reported less than five cases.

Becks, et al,[24] reported that ACTH inhibits bone growth of rats experimentally, and Applebaum and Seelig[25] reported a loss of septal bone after three months of daily injections of 2.5 mg. of cortisone to rats. Goldsmith[26] said that rats receiving 3 mg. of cortisone daily for a year showed an increase in the amount of alveolar bone and a marked narrowing of the marrow spaces. He said that the appearance may be the result of a retardation of endosteal resorption. In the periodontal membrane a disorganization of the arrangement of the fibers and their attachment to the bone was seen. Where the animals received .5 mg. of cortisone daily, no changes were seen.

Glickman, et al,[27] injected 20 mice with .5 mg. of cortisone for periods up to 43 days. Microscopic examination of the periodontal tissues revealed osteoporosis of alveolar bone characterized by reduction in the number of osteoblasts and amount of newly formed osteoid matrix; reduction of alveolar bone, edema of the periodontal membrane with reduction in the number of fibroblasts and collagen fibers, and degeneration of the collagen fibers. These changes occurred unrelated to gingival inflammation associated with local irritation.

The bone loss was attributed to a disturbance in the corticosteroid hormone balance responsible for the anabolic-catabolic equilibrium essential for the maintenance of bone.

Glickman and Shklar[28] injected a group of 20 animals with .25 mg. cortisone acetate daily, another

20 were given .25 mg. cortisone acetate and one weekly injection of 600 rat units of estradiol benzoate, an estrogenic hormone. Another group of 10 was given daily saline injections, and another group was not injected. At the end of a 35 day experiment, microscopic study showed that the systemic administration of estradiol benzoate effected a modification of the osteoporotic effect observed when cortisone is administered alone. Douglas and Kresberg,[29] however, point out that Glickman in his experiments administered a dose twenty times greater than that given to a human being when computed on the basis of milligrams per kilogram of body weight, and refer to a statement by Kerr[30] who said that there are dangers in applying the results of experiments on animals as being valid for human beings.

METHOD

The teeth and gingival tissues of 100 patients receiving significant amounts of either ACTH or one of the adreno-corticoid hormones for various systemic diseases, were examined and compared with 101 patients being treated in the same hospitals for essentially the same diseases, who were not receiving any of these drugs. The patients examined were either out-patients or bed-patients at five large hospitals. The various hospitals used ensured a variety of patients in each group from the various economic and social strata.

A rather detailed observation was made of the gingival tissues for the purpose of determining the possible effects of these drugs. The degree of inflammation, the amount of calculus, the severity of the periodontal disease as indicated by the depth and number of pockets was noted for each area. In each case, the most severe deviation from normal was noted. Where inflammatory change was evident, the depth of the associated pocket was noted as well as the number of such deviations. The inflammatory change was evaluated by noting the changes in color, form, and density of the gingival tissue. The amount of calculus was graded, and the depth of pocket formation and the state of the oral hygiene was evaluated. Other oral findings of significance such as missing teeth, rampant decay, and food impactions were used to evaluate the oral hygiene of the patient. On the basis of these observations, the nature and severity of periodontal disease was classified as follows: gingivitis (moderate or severe), or periodontitis.

The medical diagnosis for each patient, was in all cases taken from the medical charts. Other medical findings of significance, if available, were also noted. In those cases receiving steroid therapy, the name of the drug, the dosage, and the length of time taken were also listed. Observations were also made and recorded of a group of patients with similar diagnoses who were used as a control group. These patients were examined without prior knowledge as to whether or not they were receiving steroid drugs. On the basis of the examination and observations made, the type and severity of periodontal disease was listed, as well as the average amount of inflammatory change present in the mouth. Oral status was considered a composite of oral hygiene and calculus deposition, with some consideration given to "other oral findings of significance." Oral status was classified as good, fair or poor.

FINDINGS

The following charts illustrate the major findings of the examinations that were made. The figures represent the number of patients showing the various characteristics.

DISCUSSION

These observations indicate that there are far less patients with periodontitis in the steroid group than in the control group. However, 51% of the steroid patients are under the age of 41, while only 33% of the control patients are under 41. Also, 68% of the control patients have a poor oral status, but 40% are poor in the steroid group. In the control group, as the age of the patient increases, the number of periodontitis cases increases markedly. In the steroid group, while there is some increase in the number of periodontitis cases, the incidence never rises above 50%. In comparing the oral status in control and steroid groups in the age group over 40, the oral status of the steroid group is much better

CHART I

Comparison of the Nature of Periodontal Disease, Inflammatory Change, and Oral Status of the Steroid and Control Groups

Age Groups	*Diagnosis*		*Oral Status*		*Inflammation*	
	No. Steroid	*No. Control*	*No. Steroid*	*No. Control*	*No. Steroid*	*No. Control*
10-20	18 ging.	4 ging.	1 good	1 good	4+	
			15 fair	2 fair	11++	3++
			2 poor	1 poor	3+++	1+++
21-30	16 ging.	8 ging.	2 good	2 good	5+	3+
	2 perio.	2 perio.	10 fair	5 fair	6++	5++
			6 poor	3 poor	7+++	2+++
31-40	15 ging.	12 ging.	3 good	3 good	2+	6+
	9 perio.	7 perio.	10 fair	5 fair	16++	5++
			11 poor	11 poor	6+++	8+++
41-50	9 ging.	7 ging.	1 good	2 good	3+	2+
	9 perio.	17 perio.	9 fair	2 fair	9++	13++
			8 poor	20 poor	6+++	9+++
51-60	8 ging.	7 ging.	4 good	2 good	3+	3+
	7 perio.	23 perio.	2 fair	8 fair	8++	13++
			9 poor	20 poor	4+++	14+++
60-	4 ging.	4 ging.				1+
	3 perio.	10 perio.	3 fair	1 fair	5++	7++
			4 poor	13 poor	2+++	6+++
Total	70 ging.	42 ging.	11 good	10 good	17+	15+
	30 perio.	59 perio.	49 fair	23 fair	55++	46++
			40 poor	68 poor	28+++	40+++

Ging. is an abbreviation of gingivitis.
Perio. is an abbreviation of periodontitis.
+indicates that the average amount of inflammation was mild.
++indicates that the average amount of inflammation was moderate.
+++indicates that the average amount of inflammation was severe.

than that of the control group, and would account for the differences mentioned.

There is also a direct correlation between the degree of inflammation and oral status which is not dependent on therapy with steroids. 68% of the control patients have a poor oral status and 40% of the patients show severe inflammatory reactions. 40% of the steroid patients have a poor oral status and 28% show severe inflammatory reaction. In the 21-30 age group, there is a more severe inflammatory response in the steroid group as compared with the oral status, than in the control group. In the 41-50 age group, the inflammatory reaction in the control group is less severe than expected considering the large number of patients with a poor oral status. Also, in the over-60 age group, the inflammatory reaction in the control group is less severe than expected considering the oral status.

In comparing those patients receiving ACTH with those receiving drugs more closely related to the adrenal cortex, the results parallel the findings in Chart I.

The ACTH patients had a proportionately poorer oral status than the steroid group, and showed a relatively more severe inflammatory reaction. Chart II illustrates these findings.

Although the chart does not so indicate, all of the 11 patients under the age of 40 receiving ACTH therapy had a diagnosis of gingivitis, and all but two of the patients over 40 had a diagnosis of periodontitis.

CHART II

Comparison of the Nature of the Peridontal Disease, Inflammatory Change, and Oral Status in Patients Receiving ACTH and Those Receiving Steroids

	Diagnosis	*Inflammation*	*Oral Status*
ACTH	13 ging.	1+	1 good
	5 perio.	8++	5 fair
		9+++	12 poor
Steroids	57 ging.	16+	10 good
	25 perio.	47++	44 fair
		19+++	28 poor

There are far more periodontitis cases in the group of patients receiving steroids for longer periods than in those on steroids for two months or less. However, those patients receiving steroids for a longer period were generally in the older age groups. The patients receiving steroids for the shorter period had the poorer oral status and a resultant more severe inflammatory response. Charts III and IV illustrate these findings.

In patients with a diagnosis of rheumatoid arthritis, 57% of the patients receiving steroids have periodontitis, and 55% in the control group have periodontitis. 42% of the rheumatoid arthritis patients in the steroid group have a poor oral status and 24% have an inflammatory reaction classified as severe. 63% of the rheumatoid arthritis patients in the control group have a poor oral status and 35% have an inflammatory reaction classified as severe. In general, the findings concur with the overall observations made and listed in Chart I. The nature of the periodontal disease, and the amount of inflammatory change are more dependent on age and oral status than on steroid therapy.

In a fairly large group of control patients with a diagnosis of osteo-arthritis, there are proportionately more patients with a poor oral status, more patients with severe inflammatory reactions, and more patients with periodontitis. In a group of steroid patients with eye disease there are large numbers of patients with a poor oral status, a more severe inflammatory reaction as a result, and relatively fewer periodontitis cases because this was a younger age group.

A consideration of any other categories of disease within the control and steroid groups do not contain significant differences.

CHART III

Comparison of the Nature of Peridontal Disease, Inflammatory Change, and Oral Status in Patients Receiving Steroids for Varying Intervals

Dosage Interval	*Diagnosis*	*Inflammation*	*Oral Status*
1 week-2 months	46 ging.	5+	4 good
	15 perio.	33++	31 fair
		23+++	26 poor
3 months-9 months	12 ging.	7+	4 good
	4 perio.	7++	6 fair
		2+++	6 poor
11 months-6 years	12 ging.	5+	3 good
	11 perio.	15++	13 fair
		3+++	7 poor

CHART IV

Age Ranges of Patients Receiving Steroids for Varying Intervals

Age	*Dosage Interval 1 week-2 months*	*Dosage Interval 3 months-6 years*
10-20	11	7
21-30	14	4
31-40	15	9
41-50	10	8
51-60	4	10
60-	7	1

SUMMARY

The gingival tissues of 100 patients receiving steroid drugs as bed patients in various hospitals or in several out-patient departments were examined and compared with the gingival tissues of 101 patients from the same hospitals not receiving these drugs. A rather detailed analysis was made of all of these patients noting age, inflammatory changes, type and severity of periodontal disease, and local factors which might affect the gingival tissues. The medical diagnosis was noted, as well as the dos-

age and time interval of steroid therapy, if any.

It was found that in both the control and steroid groups, as the oral status of the patient became poorer, the inflammatory changes became more severe. Also, the nature of the periodontal disease grew more severe as the age of the patients in each of the groups increased.

CONCLUSIONS

1. The extent of the inflammatory response of the gingival tissues as well as the diagnosis of periodontal disease were more dependent upon the age and oral status of the individual than on whether or not the patient had been receiving steroids.

2. Steroid therapy does not produce any gingival reaction.

3. No significant differences in gingival tissue can be seen between the effect of ACTH and the steroids associated with the adrenal cortex.

4. When the patients receiving steroids are divided into groups who have had the drug for two months or less and those receiving it for a longer period of time, the findings are correlated with the age and oral status.

5. In comparing patients in the steroid group and those in the control group with rheumatoid arthritis as their prime reason for medical treatment, no significant differences can be seen.

6. In studying a control group of patients with osteo-arthritis, and a steroid group with eye disease, the periodontal disease evident can be explained on the basis of age and oral status.

7. Other categories of diseases present in this study do not demonstrate any significant differences.

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Announcements . . . .

SPECIAL RESEARCH COMMITTEE APPOINTED BY N.I.D.R.

Formation of a special committee to aid in developing a broadened research attack upon the oral health problem of pyorrhea was announced today by Dr. F. A. Arnold, Jr., Director of the National Institute of Dental Research, one of the Public Health Service's National Institutes of Health.

Immediate plans will be made for an accelerated effort to combat periodontal disease, which affects a large proportion of the Nation's older citizens. In developing the broadened program, this special committee will work with the Institute and the National Advisory Dental Research Council, established by the National Dental Research Act in 1948 to advise upon dental research and related activities.

Members of the Committee are:

Dr. Edgar D. Coolidge, Professor Emeritus, Loyola University, Chicago (Chairman).

Dr. Lucian A. Bavetta, Professor of Biochemistry and Nutrition, University of Southern California, Los Angeles.

Dr. Frank E. Beube, Head, Division of Periodontology, School of Dentistry and Oral Pathology, Columbia University, New York City.

Dr. Henry M. Goldman, Chairman, Department of Periodontology, University of Pennsylvania and Director, Riesman Dental Clinic, Beth Israel Hospital, Boston.

Dr. Harold C. Ray, Chairman, Division of Periodontology, University of California, San Francisco.

Dr. Helmut A. Zander, Head, Department of Periodontology, Eastman Dental Dispensary, Rochester, New York.

Under the current program of the National Institute of Dental Research, some 36 research projects in the field of pyorrhea have been recommended by the National Advisory Dental Research Council for support in the amount of about $500,000. These 36 research projects are to investigate the causes, methods of treatment, and prevention of pyorrhea and its effects which lead to impaired mastication and the need for dentures or partial prosthetic devices.

The Dental Research Council is one of nine such Councils, established by law as advisors to the Surgeon General of the PHS for the programs of NIH. Comprised

chiefly of non-Federal scientists, educators, and civic affairs leaders, the groups recommend upon grants for research, and these awards can be made by the Surgeon General only after Council recommendations.

ROWE SMITH FOUNDATION

The Rowe Smith Memorial Foundation announces its Fifth Annual Seminar to be held January 18-21, 1959. The subject will be "The Blood and Bleeding." Moderator will be Dr. E. Cheraskin and Essayists Dr. Joseph M. Hill, Dr. Raymond W. Monto, and Dr. Robert B. Shira. For further information please contact Rowe Smith Memorial Foundation, Inc., 501 State National Bank Building, Texarkana, Ark.-Texas.

ASSOCIATION OF AMERICAN WOMEN DENTISTS

The Association of American Women Dentists annual meeting will be held in Dallas, Texas on Monday, November 10th, 1958. Headquarters will be the Baker Hotel and they will be open on Sunday the 9th for visiting women dentists.

DR. C. WESLEY MARRIOTT

March 30, 1887—August 30, 1958

Dr. C. Wesley Marriott, well known dentist of Cleveland, Ohio died in the Veterans Hospital in Minneapolis on August 30, 1958. Although he retired from active practice in 1949 because of ill health, he still has many friends in the Academy of Periodontology as he had been a member for twenty-five years. He served very capably as treasurer of the Academy for several years, and served well on several committees. The Academy—and all dentistry—is indebted to Dr. Marriott for his faithful services.

Book Reviews . . . .

Periodontics. Balint Orban. Editorial Staff: Frank M. Wentz, Frank G. Everett, and Daniel A. Grant. *The C. V. Mosby Company, St. Louis, 1958. 537 pages and 439 illustrations. Price $14.50.*

This book is written in the spirit and tradition of the great "Vienna School," and most appropriately, it is dedicated to the memory of Dr. Gottlieb. The impact on periodontics by the eminent and dynamic scientists and teachers from Vienna can be observed over the entire world. The leading position of today's American periodontics is to a great extent the result of a fortunate synthesis between the histopathologic approach of this "Vienna School" and the highly developed technical skill of American dentistry.

This new textbook by Orban and co-authors follows very closely the basic philosophy of Gottlieb and Orban's "Zahnfleischentzündung und Zahnlocherung" first published in 1933, and later in 1938 translated to the English with some alterations and additions. A tremendous scientific and clinical development has taken place in periodontics since 1938, and the new book has been brought fully up to date on biologic principles as well as on technical aspects of periodontics. For example, the most promising research in periodontics today is probably within the field of oral microbiology and enzymology, and an outstanding review of this field is presented.

The controversial subject of trauma from occlusion has received extensive coverage (about one fourth of the book). Histopathologic findings associated with experimental trauma are described and illustrated in great detail. Clinical procedures for occlusal adjustment are well illustrated and mostly based on generally accepted principles. A few controversial statements are unavoidable when attempts are made to cover the field of occlusion.

The entire book is characterized by a "middle of the road" cautious approach to treatment procedures. On page 235, for example, it is stated: "It would be as great an error to try to make every case fit a preconceived idea of gingival form as it would be to make every case fit a preconceived idea of occlusion." Clinical procedures are always evaluated in relation to rational biologic principles and clinical observation over a long period of time.

In the commendable attempt to make the textbook as brief and concise as possible, it appears that some important parts of periodontics have been "shortchanged." Epidemiology and prevention of periodontal disease, for example, is given only a page and a half; and the chapter on orthodontic measures and splinting is mainly an accumulation of case reports without the adequate discussion of biologic principles which characterizes other parts of the book.

The illustrations are descriptive, technically of first class, and the reproduction is excellent. The reproduction of roentgenograms is the best this reviewer has ever seen.

In the preface it is stated: "This text constitutes background material for teaching the basic principles of periodontics on which sound practical methods can be based." This goal has been reached within the framework of 537 well-written, well-organized, and well-printed pages. The result is a beautiful, stimulating, and informative textbook that will be read over and over again by everybody who is interested in periodontics.

Should somebody by chance look into the book without previous interest in periodontics, he also will run a high risk of being "caught" and converted to periodontics by this inspiring book. *Sigurd Ramfjord, L.D.S., Ph.D.*

Clinical Periodontology

Recognition, Diagnosis and Treatment of Periodontal Disease in the Practice of General Dentistry, Irving Glickman, *2nd Edition. 978 pages with 720 figures. W. B. Saunders Company, Philadelphia, 1958. $17.50.*

The preface to this second edition reiterates the author's "firm conviction that the periodontal care of the American public is primarily the responsibility of the general practitioner. . . ." The present edition has been prepared to provide the general practitioner and the dental student with a comprehensive text presenting material in such a manner that it can be used in daily chairside practice. The preface also states that "this remains a textbook written by a teacher for those who want to learn effective, uncomplicated methods of treating periodontal disease. . . ."

The book is divided into three sections. Section one on The Tissues of the Periodontium covers 60 pages. Section two on Periodontal Disease is divided into four parts covering 390 pages. Section three on the Clinical Management of Periodontal Disease consists of five parts covering 494 pages. It is useful to note that the usual repetitious, but undoubtedly necessary, review of oral histology is kept to a minimum and that almost 900 pages of the text are devoted to diagnostic and treatment procedures. A comprehensive index is included and excellent lists of references are found with each chapter.

The technical details of the book are excellent. The author writes well and the illustrations are very striking. There are two colored plates which probably serve no useful purpose since they are obviously drawings instead of photographs. Some of the procedures are illustrated with photographs and others are excellently illustrated with drawings. This latter seems a valid procedure but it might be better if no drawings of final results were included. These are too ideal to be realistic.

Criticisms are too often the outgrowth of personal preferences. Some of this sort might be offered here. For example, several of the illustrations would be more authoritative if photographs had been used instead of drawings, no matter how artistically the drawings are made. One chapter is devoted to classification, in which the existing confusion is well demonstrated; and then the author compounds this confusion by offering still another classification. It would seem that the authors of textbooks could bring themselves to accept the recommendations of the Committee on Nomenclature and Classification of The American Academy of Periodontology.

This book is written for the student and for the general practitioner who wants a guide for a beginning periodontal practice in a general office. It accomplishes this purpose admirably and is a major contribution to the modern dental literature. It can be highly recommended as a text and reference book. *C. Kenneth Collings.*

Periodontology, by Edgar D. Coolidge and Maynard K. Hine. *Third edition. 429 pages; 463 illustrations, 268 figures and 2 color plates. Lea & Febiger, Philadelphia. 1958. Price: $7.50.*

The new third edition of the book by Coolidge and Hine has been revised and expanded and given a more appropriate title, "Periodontology." Although this latest edition shows considerable resemblance to the preceding one, sections have been rearranged and rewritten, and new material added.

Two new chapters in the third edition have widened the scope of coverage. The first of these, *Halitosis and its Treatment*, considers the sense of smell, odors, and halitosis in particular. It is highly informative and fascinatingly written. The other chapter, *Nutrition and Periodontal Disease*, presents a cursory review of some of the more important and well established aspects of nutrition. Its value lies in presenting in a concise and rational manner the role that nutrition plays in the etiology and control of periodontal and oral disturbances. Although some of the finer and more subtle effects of nutrition have been left out, the chapter does give an insight into this important field.

This text reflects the excellent academic background of the authors. Emphasis is placed upon the presentation and utilization of biologic knowledge in clinical treatment rather than upon description of technics of treatment. The impression gained is that the authors are primarily interested in establishing a sound therapeutic philosophy.

This textbook is especially recommended for the undergraduate dental student and for the dentist who seeks a more profound grasp of periodontal problems. The periodontist who is already well grounded or the advanced student in periodontics is apt to find this textbook insufficiently detailed in the clinical phase of treatment.

As a whole, the textbook is well organized, well written, and clearly illustrated. Although it gives broad coverage of the field of periodontology it does this in a clear and concise manner. *Charles G. Maurice, D.D.S.*

Oral Hygiene, Bunting, Russell W., *Lea & Febiger, Philadelphia, 1957. 334 pages, 233 illustrations and 1 color plate. 3rd edition. $7.00.*

Although the title of the book is Oral Hygiene, the coverage of the subject is in the broader sense which includes all those conditions pertaining to the health of the mouth and its preservation. Beginning with a delineation of the fundamental principles of preventive dentistry, subsequent chapters are devoted to correlated and related information pertaining to the anatomy, histology and embryology, and the physiology of the oral structures. These chapters are followed by those dealing with the etiology of the pathological factors which attack the hard and soft tissues. Methods by which diseases may be prevented or controlled are then presented and include techniques of dental prophylaxis. Each chapter is written by an outstanding dental scientist who has made contributions to the specific field.

Dr. Bunting has dedicated the three editions of his book to Dr. Arthur C. Fones who is recognized as one of the first to bring the subject of the hygiene of the mouth to the attention of the dental profession. Dr. Fones predicted the value and scope of the auxiliary services of the dental hygienist. That his early prediction has now become a reality is evidenced in two chapters written by dental hygienists on the dental hygienist in public health and the history and organization of dental hygiene.

While one will recognize some of the content and illustrations as being identical to what has appeared in previous editions, the chapters have been revised and amplified, a new chapter has been added on the pathology of dental caries and one hundred additional illustrations have been included.

It was the intention of the author to direct the text to the needs of dental hygiene education by presenting specific information not included in standard textbooks. However, the use of the book is not limited to dental hygienists. Since it contains information relative to source material in oral hygiene and preventive dentistry, it is also useful for teachers who wish to plan teaching units on dental health and to members of the dental profession who wish to participate in dental health education programs.

A. Rebekah Fisk

Periodontia. By Henry M. Goldman, D.M.D., F.A.C.D., and D. Walter Cohen, D.D.S. *Fourth Edition, 535 pages with 366 illustrations. $12.50. St. Louis, Missouri, C. V. Mosby Company, 1957.*

The fourth edition of this textbook on Periodontia should be considered as an entirely different text from the previous editions. The authors have completely eliminated therapy and concentrated on principles and procedures of examination and diagnosis of periodontal disease as well as their etiology. The treatment phase of periodontal diseases is discussed in another text by the senior author with Schluger and Fox.

This text considers the structures of the periodontium from an anatomical, histological, physiological basis and supplements this information with radiological examination. Oral Physiology is discussed followed by pathology of the periodontium including variations in function.

A chapter is devoted to the various investigations to solve periodontal diseases through research. The remaining chapters discuss the etiology and diagnosis of the many involvements of the periodontium. These are well supplemented with excellent photographs and roentgenograms.

The text throughout is well written, concise, and easily understood. The student, general practitioner, and periodontist will find this book a valuable addition to his library.

Henry M. Swenson

The Gateway to Health by Ira Jay Berlove, D.D.S., M.D., *141 pages, 9 text illustrations. Price $3.50. New York. Whither Books, Inc. 1958.*

This book contains simple though accurate descriptions of dental disease, oral hygiene and explains the relationship of oral with general health. The author, Dr. Berlove, is a dentist, a physician and has devoted many years to the specialty dealing with diseases of the nose and throat. This background makes Dr. Berlove's words especially helpful not only as far as dentistry is concerned but also to the closely adjacent regions of the nose, throat and sinuses.

The book is written for the layman so that he can be intelligently informed on the dental problems with which he is faced. He will learn how these problems can be sensibly solved. Moreover, the book is written in such a sprightly fashion that although it is a thorough discussion of dental and other maladies it makes understandable enjoyable reading. Every dentist should have available copies of this book to loan to his patients to help them understand their dental problems. There are twenty-one chapters covering in clear words everything from dentifrices to full dentures. *Grant Van Huysen*

A TEXTBOOK OF ORAL PATHOLOGY

By William G. Shafer, B.S., D.D.S., M.S., Maynard K. Hine, D.D.S., M.S., and Barnet M. Levy, A.B., D.D.S., M.S., *714 pages and 416 text illustrations, W. B. Saunders Co., Philadelphia and London, 1958. $15.00.*

A textbook of Oral Pathology represents a stringent, concise compilation of oral diseases, both common and uncommon, with considerable emphasis placed on clinical and radiographic features in addition to the usual histopathologic treatment. Clinical photos and photomicrographs have been carefully selected to demonstrate salient features without laborious repetition of inconsequential detail. There is a refreshing lack of reference to monotonous case histories or personal experiences. Treatment and prognosis is discussed briefly and succinctly and without embellishment.

As stated by the authors in the preface, an attempt has been made "to integrate information from many of the basic sciences for adequate diagnosis of oral disease." Certainly, from the student's point of view, the transition from general pathology to oral pathology is readily accomplished. Perhaps this is a function of the format of the book which is divided into 5 sections: "Disturbances of Development and Growth" (which includes tumors), "Diseases of Microbial Origin" (including dental caries and pulp infections), "Injuries and Repair," "Disturbances of Metabolism" (which treats of nutritional and endocrine relationships to oral disease), and "Diseases of Specific Systems" (which includes diseases of bone and joints, blood and blood-forming organs, the periodontium, skin and nerves). Of particular value, although apparently unique in most pathology texts, is the addition of appendix tables of normal laboratory values, blood and urine values, normal nutritional requirements and normal chronologic tables of tooth development.

This is a book which could function equally well as a text for the dental student or as a practical reference for the medical or dental practitioner. *S. M. Standish.*

Announcements . . . .

ARMED FORCES INSTITUTE OF PATHOLOGY

During the period March 23-27, 1959 the Oral Pathology Branch, Armed Forces Institute of Pathology will present its annual course on "Pathology of the Oral Regions." This course is open to both government and civilian dentists. Dentists in the federal establishment should apply through appropriate channels and civilian dentists to the address below. Information as to course content will be given in a later announcement. There is no tuition and applications should be made as soon as possible because of heavy registration.

Slide Seminar on "Advanced Oral Pathology"

The annual slide seminar conducted by the Oral Pathology Branch of the Armed Forces Institute of Pathology will be on May 22, 1959. Approximately 60 can be accommodated. A slide set of 25 slides together with a descriptive brochure will be given each one attending for his permanent possession.

This is a seminar on Advanced Oral Pathology and the specific subject will be "Tumors of the Maxillary and Mandibular Bones." Teachers and workers in Oral Pathology, Oral Diagnosis and Oral Medicine will be given preference on the registration. There is no charge for this seminar.

Those desiring to register or wishing information regarding these courses should write:

Colonel Joseph L. Bernier
Chief, Oral Pathology Branch
Armed Forces Institute of Pathology
Washington 25, D.C.

WESTERN RESERVE UNIVERSITY

The Division of Postgraduate Education of the School of Dentistry of Western Reserve University announces that Drs. Morton Amsterdam and Walter Cohen of the staff of the School of Dentistry of the University of Pennsylvania will present a course on Periodontal Prosthesis January 3, 4 and 5, 1959. The fee for this course is $75.00. Those interested should write Dr. Philip Burwasser, Director of Postgraduate Education, School of Dentistry, Western Reserve University, Cleveland 6, Ohio.

DR. SAMUEL CHARLES MILLER MEMORIAL LECTURE

The first Dr. Samuel Charles Miller Memorial lecture will take place on Tuesday, February 10, 1959 at 8:00 p.m. in the new building of the New York University College of Dentistry located at 421 First Ave., New York 10, N. Y. The entire dental profession is cordially invited to attend.

Sidney Sorrin, Chairman

THE AMERICAN ACADEMY OF ORAL PATHOLOGY

The American Academy of Oral Pathology announces that the examination for elevation to Fellowship in the Academy will be held April 17, 1959 at the University of Minnesota, Minneapolis, Minnesota. Applications for the Fellowship Examination may be obtained from Dr. Robert J. Gorlin, Secretary-treasurer, American Academy of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis 14, Minnesota. No applications will be accepted after January 1, 1959.

UNIVERSITY OF ALABAMA

During the month of January the University of Alabama School of Dentistry will present the following refresher courses: *The Recognition, Identification & Treatment of Oral Disease* will be conducted by Colonel Joseph L. Bernier, DC, USA on January 10, 11, and 12; *High Speed Techniques* will be offered on January 16, 17, and 18 by Adeeb E. Thomas, D.M.D.; *Saul Schluger,* D.D.S. will instruct a course in *Periodontology for the General Practitioner* on January 24, 25, and 26; and Arthur H. Wuehrmann, D.M.D. will conduct a course in *Oral Roentgenology* on January 30, 31, and February 1.

For further information, please write to the Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Avenue South, Birmingham, Alabama.

UNIVERSITY OF WASHINGTON

The University of Washington School of Dentistry offers a new course of graduate study in Periodontics. The course is of eighteen months duration and upon the satisfactory completion of a thesis, in addition to the acceptable performance of the remainder of the academic program, leads to the granting of a Master of Science degree in Dentistry.

The course will be directed toward the training of teachers and research workers in Periodontics and allied fields. Extensive clinical training will be offered in addition to the projection of a basic science background.

Inquiries should be addressed to: Saul Schluger, D.D.S., Director Graduate Dental Education, University of Washington, School of Dentistry, Seattle 5, Washington.

BOSTON UNIVERSITY SCHOOL OF MEDICINE POST GRADUATE COURSES

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University, wishes to announce the dates of the following courses.

PERIODONTAL THERAPY	OCTOBER 6-17	1958
PERIODONTAL PATHOLOGY	OCTOBER 27-NOVEMBER 1	1958
ENDODONTICS	NOVEMBER 10-14	1958
OCCLUSION AND OCCLUSAL ADJUSTMENT	NOVEMBER 10-14	1958
ORAL PATHOLOGY (For Oral Surgeons)	JANUARY 12-16	1959

Those interested may write to the Department of Stomatology, Division of Graduate Studies, School of Medicine, Office of the Dean, Boston University, 80 East Concord Street, Boston 18, Massachusetts.

CUBAN DENTAL CONGRESS

The V Congreso Dental Nacional and I Congreso Interamericano Dental de Cuba, will be held next March 29, 30 and 31st of 1959 in Habana. For information Colegio Estomatológico Nacional, calle L No. 353, Vedado, Habana, Cuba.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Tufts University School of Dental Medicine announces the following Postgraduate Refresher Courses:

DPG. 602—Occlusal Equilibration

February 9, 10, 11, 1959 Tuition $100.00—Class limited to 12

This is a practical course presented by Dr. Edward T. Fischer, Assistant Professor of Periodontology devoted to the detection and correction of trauma from occlusion. It is a course for the general practitioner, the periodontist, the orthodontist and the prosthodontist. Technics for detecting occlusal disharmonies will be analyzed and demonstrated on patients. The technic for equilibrating and occlusion will be described.

DPG. 603—Occlusal Adjustment

March 30, 31, April 1, 1959 Tuition $100.00—Class limited to 12

Dr. Ramfjord, Associate Professor of Periodontics, University of Michigan School of Dentistry has been interested in occlusal problems since 1937, and as a result he has developed a teaching program of considerable practical value to the dental practitioner. The course includes seminar type lectures and demonstrations on patients. The lectures cover, among other topics, the principles of occlusion and the rules and procedures for occlusal adjustment. Clinical demonstrations include the recording of centric relation and the detection of occlusal interferences in centric as well as in lateral and protrusive excursions.

For additional information please write Division of Graduate and Postgraduate Studies, 136 Harrison Avenue, Boston, Massachusetts.

BETH ISRAEL HOSPITAL

The Dental Department of the Beth Israel Hospital announces a postgraduate course in Periodontia to be given in the Fall of 1958.

A two-week seminar in Periodontia is scheduled for October 6 to October 17. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations, and clinics. The tuition fee is $350.00. A deposit of $150.00 is necessary for enrollment.

Co-Directors of the course: Henry M. Goldman, D.M.D., Bernard Chaikin, D.M.D.

For further information, write to: Director of Public Relations, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Massachusetts.

Letters to the Editor . . . .

To the Editor:

I am writing to discuss an article, Reattachment and Roentgenograms by Dr. Nathan Friedman which appeared in the April, 1958 issue of the Journal.

In this article Dr. Friedman criticizes the use of roentgenographic evidence to support claims of reattachment in the treatment of periodontal disease, the basis of which claims is the assumption that increased density of bone is taken as evidence of both reattachment and bone regeneration. He states that while remaining bone becomes more dense after treatment it does not advance coronally beyond its previous highest level.

Dr. Friedman further criticizes the use of probes to prove reattachment, stating that evidence so gained is highly subjective. He indicates that evaluation of evidence so gained is influenced by the bulk of the probe used. He feels, further, that histological evidence of reattachment has in the main been unsatisfactory.

I believe that the foregoing summarizes fairly Dr. Friedman's objections to the use of roentgenographic evidence regarding reattachment. In discussing his article let me first state points in which I agree with him.

I agree that proof of reattachment when claimed through use of a probe is open to question, which makes all the more important the evidence of coronal bone regeneration as seen in the roentgenogram. With regard to the probe, the desire of the operator to believe that he has secured reattachment is undoubted and may well color his estimate of his results. This is all very well on a short term basis, but the periodontist who sees his patients routinely over a period of many years must face recurrences of disease if he has failed to secure real reattachment. I feel that the probe, if of proper size and used properly, will demonstrate the true state of affairs.

More valid is Dr. Friedman's criticism of the bulk of probes often used and illustrated in articles claiming reattachment. Penetration of a bulky probe into a treated pocket may well be blocked by close apposition to the root of healed but not necessarily reattached tissue. I personally use a probe made by breaking off the tip of a No. 17 explorer, thus reducing it to a bayonet shape. This provides the slenderest practicable probe. Pockets which refuse to admit this probe to their former depth over a period of years can

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safely be considered to have a reattachment. I might add that for roentgenographic purposes I use a slender steel orthodontic ligature wire as a probe.

Histological evidence that both connective tissue and epithelial reattachment can take place was first adduced by Box in 1924. Unfortunately, Dr. Friedman apparently had not seen this early publication. The later one to which he refers does not give quite such conclusive evidence. In 1926 I presented a method for inducing reattachment and reproduced one of Box's illustrations. Dr. Friedman mentions my article regarding the method described but apparently overlooked the illustration noted.

With regard to evaluating roentgenographic evidence of bone regeneration (coronal extension, not merely increased density of present bone) I must take issue with Dr. Friedman in that he has, inadvertently I am sure, overlooked evidence of such regeneration which I believe cannot be discounted. I refer to case reports by Bjorndahl in 1948 and myself in 1951, both published in the Journal of the American Dental Association. He also evidently failed to see Cross's first article on bone regeneration published in an English journal, Dental Practitioner (1955); evidence adduced in that article is more conclusive than the evidence in the article cited by Dr. Friedman.

One more item must be mentioned:—the type of operation chosen for periodontal treatment. Dr. Friedman, although not describing it in detail, evidently has resorted to gingivectomy and osteoplasty in the cases he presented. It seems obvious to me that while some reattachment may take place in cases so treated, regeneration of bone in a coronal direction cannot be expected.

The method I have followed in the majority of cases for many years and first presented in 1926 (more recently, with modification of formula, in 1952) is spoken of as the conservative method. It provides maximum retention of gingival tissue, consonant with expected opportunity of reattachment, bone is seldom curetted, hence there is the maximum opportunity for regeneration. As to roentgenographic evidence of bone regeneration in a coronal direction. I may add that men whom I regard as "top" periodontists have accepted this evidence and my claims.

In view of the undoubted possibilities for attaining reattachment and bone regeneration I think it is highly unfortunate for a periodontist of Dr. Friedman's ability and standing to take the negative attitude expressed in his article.

John Oppie McCall, D.D.S.
944 Oak Park Ave.
New York, New York

To the Editor:

A recent report by Dr. Joseph C. Muhler, "Effect on the Gingiva and Occurrence of Pigmentation on the Teeth Following the Topical Application of Stannous Fluoride or Stannous Chlorofluoride" in the JOURNAL OF PERIODONTOLOGY 28:281-286, 1957, could mislead readers because it slights the effect of tin on the teeth of subjects with periodontal disease. In the adult, of course, recession oftentimes results in an exposure of cementum and it is here that the greatest danger of staining by tin ions exists. The results of my *in vivo* researches together with the *in vitro* findings of Isaacs and Brudevold ("Discoloration of Teeth by Metallic Ions," Isaacs, S. and Brudevold, F., *J. Dent. Res.*, 36:753-758, 1957) clearly raise the possibility that prolonged clinical exposure to stannous ion may stain the necks of teeth. Further, in a recent paper of his

own "Effect of Topically Applied Stannous Chlorofluoride on the Dental Caries Experience in Children," Howell, C. L. and Muhler, J. C., *J.A.D.A.* 55:493-495, 1957, Dr. Muhler recognizes the reactivity of tin with the teeth by pointing out that the stannous ion has a high "affinity for the enamel surface even when present in micro concentrations." Incidentally, the study referred to above should be considered as an *in vivo* study since the research described was done on teeth in situ. In vitro studies of teeth exposed to 4% stannous fluoride, and *no* hydrogen sulfide solution, but exposed to a saliva and protein mixture (meat or eggs) exhibited very marked staining—especially of root surfaces.

Since discoloration of the necks of teeth as well as the crowns is an important consideration in clinical practice, it would seem only reasonable to suggest that widespread use of stannous fluoride or other tin compounds be discouraged until it has been proven beyond reasonable doubt that no objectionable staining will result after years of use.

Max Shapiro

To the Editor:

Thank you for allowing me to read the letter from Dr. Shapiro and to comment upon it. A careful rereading of my paper will indicate that the main object of the paper was to consider the effect of tin on caries arrestment and on the gingiva.

We have in our files several thousand letters from dentists all over the world who use stannous fluoride topically for both children and adults up to the age of 87 years and have failed to notice any adverse effects from the stannous fluoride solution. In addition, there are two Armed Services of the U.S. Dept. of Defense who are considering making it mandatory for every service personnel to have at least one topical application of stannous fluoride per year.

It is true that Dr. Shapiro reported on tests made *in situ*. However, he used a solution (saturated solution of hydrogen sulfide) which would never occur physiologically in the mouth. In other words, his experiment was unrealistic.

Obviously we would not recommend the use of any solution which would be harmful and one which was not properly evaluated beforehand. The beneficial effects of stannous fluoride seem so desirable that its use seems justified. It should also be pointed out that thousands of people each day are receiving stannous fluoride and we have less than a handful of complaints concerning any adverse effects. We do see, however, results which suggest strongly that stannous fluoride is a highly effective adjunct to the dentist's preventive dentistry program.

Joseph C. Muhler
1121 W. Michigan
Indianapolis, Indiana

To the Editor:

The article, "Thoughts Concerning Cause, Pathogenesis, Treatment and Prevention of Periodontal Disease" by Sumter Arnim and appearing in the July issue of the Journal of Periodontology has given me cause to write a few remarks.

It seems to me that Arnim (backed seemingly by quite a number of associates) thinks that all periodontal disease may be classified as "periodontitis." I enclose here four roentgenograms showing the first molars of an individual twenty-two years of age. Does Arnim recognize this disturbance as a "Periodontal Disease"? I would surely like to have his explanation for the development of this condition based on the thoughts expressed in the aforementioned article!

According to Arnim's thoughts, periodontal disease starts quite early—"The pathogenesis of periodontal disease begins at the time the tooth erupts";—a very stimulating thought—as if we might say, "the pathogenesis of death begins at birth." Quite an idea

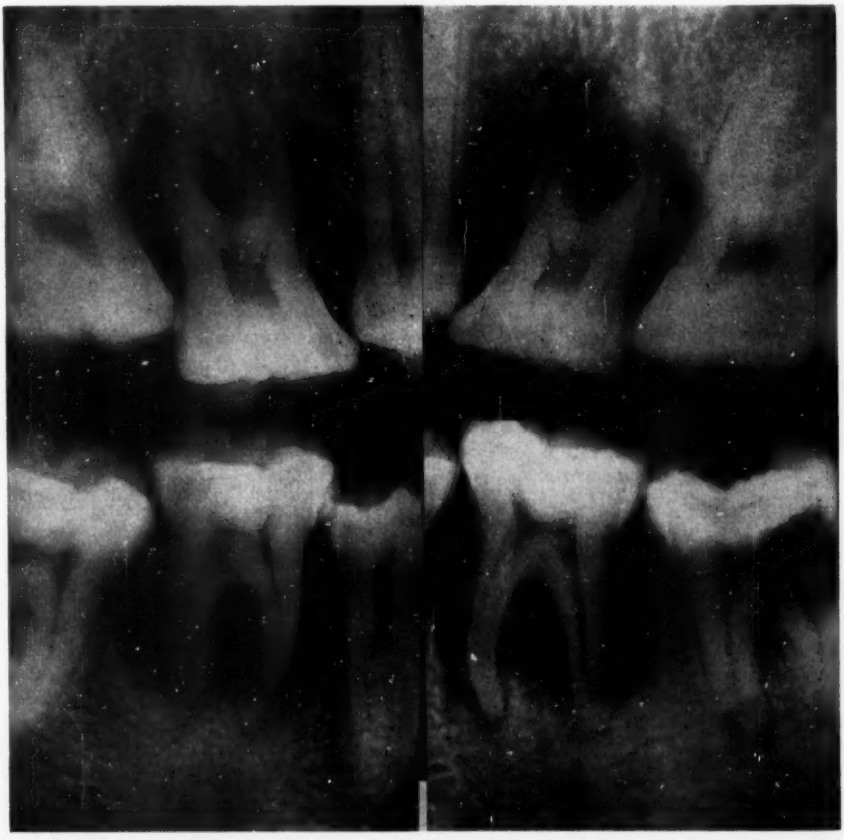

Fig. 1. Roentgenograms showing extensive bone resorption around the roots of the first molars in the dentition of a 22 year old wh. m. No other areas of resorption were present in any other region. (Courtesy Capt. Th. R. Orban, Fort Carson, Colo.)

for a research project. However, if this research project might seem to be a little vague (and no agency would risk a grant for it), why doesn't Arnim do something to prove the following statement from his paper—"These connective tissues with the serous exudate and living leukocytes provide a physiologic seal which allows for limited movements of the gingiva." Arnim uses no facts, but one quotation to substantiate his statement. May I quote from Leonardo DaVinci who said, "He who argues by citing an authority is not employing intelligence but rather memory."

The thoughts of Arnim classified according to the initials of "S", "E" and "T" sound almost like a TV ad!

I want to close by telling an old story. Once a young man read a paper in a learned society. After he finished, one of the long bearded old members of the society arose to speak and said, "Young man, your presentation contained a lot of good and a lot of new ideas. However, what is good is not new and what is new is not good!"

These are only a few thoughts stimulated by Arnim's paper!

B. Orban
933 Republic Bldg.
Denver, Colorado

To the Editor:

Illness has prevented an earlier rebuttal.

Orban and Manella (J. Perio. 27:120 April 1956) state "the deliberate removal of extensive amounts of tooth structure sometimes advocated cannot be justified," with two references to my studies (J. Perio. 23:41 1952; ibid 23:170 1952). To complete the record, I have advocated "deliberate removal of extensive amounts" in three articles since (J. Perio. 24:232 1953; ibid 25:138 1954; ibid 27:102 1956).

They say "The tooth surface should not be injured by instrumentation" (p. 132), but "the root surfaces must be completely divested of all calcareous deposits and made perfectly smooth" (p. 120).

That is the point. I have *proven* we cannot curet cementum "perfectly smooth" with a sharp instrument. They say "do it" without telling how.

Rather, they offer three illustrations which prove my contention (Figures 24-26) and blame the *type* of instrument used.

Roughening, gouging (their Figure 25), complete removal of cementum (their Figure 24), and cutting into the *softened* dentin (their Figure 26) are the usual results of curettage with a sharp instrument. Most dentists and hygienists do this. My predecessor did, from 1914 on; other practitioners have, too. I've seen their patients.

I observed this, clinically, twenty years ago; started my studies in an effort to teach my fingers *not* to gouge; found I couldn't plane to smoothness without; tried "radical curettage" in the mouth; discovered the periodontium, the patient, and I, liked it so well we have used it exclusively for some fifteen years since (no prophylaxes, no toothbrush drills, no surgery, no occlusal equilibration, no diet). We have no desire or need to return to these usually accepted procedures.

Somewhere along the line in periodontology's history (probably Hartzell) we became obsessed with the sacredness of cementum. Why? Epithelium "clings" to (not *attaches* as Gottlieb and Orban would have us believe (Waerhaug—The Gingival Pocket)) and connective tissue unites with dentin (too many references to be quoted here). Exposed dentin is not necessarily sensitive, or particularly susceptible to caries (occlusal abrasions, wedge-shaped cervical abrasions, artificial V-shaped abrasions after radical curettage, teeth from which old crowns have been removed). Such dentin should be (is, in our experience) a better outer surface than a thin layer of dead bone (cementum) or a porous layer of poorly-calcified tooth (the granular layer of Tomes). Newer studies (unpublished) seem to show that tubules become blocked and clinically eburnated, shiny, and immune to decay, like wedge-shaped cervical abrasions. Adjoining gingivae are usually healthy, whether the softened dentin has been removed by a toothbrush or a sharp instrument; inadvertently or intentionally. Look at the successfully-treated places in your own practice—the cementum and part of the dentin will be gone. If the gingivae are so thick and the pocket so deep this can't be seen, your treatment has failed.

We cannot progress if we persist in believing what we have been taught long after it has been shown wrong. I will welcome *proof* that I am in error. Until then, I lament popular opinions which may impede our growth.

A copy is being sent to Dr. Orban by the same post.

A. B. Riffle, D.D.S.
115 Monroe Parkway
Rochester 18, N. Y.

To the Editor:

I received a "Letter to the Editor" from Dr. A. B. Riffle and my answer is "No Comment."

Balint Orban, D.D.S.
933 Republic Bldg.
Denver 2, Colorado

To the Editor:

The term gingival repositioning has been used in the past to describe the plastic surgical procedure in which gingiva and mucosa is transposed apically in order to extend the zone of attached gingiva and/or increase vestibular height. The new term mucogingivoplasty is recommended to better describe the above procedure.

I also feel impelled to comment on the article "Repositioning and Increasing the Zone of Attached Gingiva" by A. A. Ariaudo and H. A. Tyrrell published in the April 1957 issue of the Journal of Periodontology.[1] Drs. Ariaudo and Tyrrell advocated vertical incisions at both ends of mucogingivoplasty flaps. This is particularly good when a single tooth requires mucogingivoplasty, for it allows us to restrict the mucogingival procedure to a small localized area. There are, however, other points in the above mentioned article that should be considered in more detail. The following paragraph is quoted from the article:

"Previously[2] a technique was described for the management of these cases. However, probable problems still existed after such treatment. These were 'shelving,' with insufficient attached gingiva present to permit corrective gingivoplasty, residual pockets at the

areas most distal to the incision and resultant narrow attached gingiva which hindered correct toothbrush massage."

The following statements are offered for consideration: If the apical transposition of the gingiva is sufficient, then there should not be residual pockets. If osseous surgery is utilized, there should not be shelving, for in mucogingivoplasty, the healed soft tissue usually reflects the contours of the underlying bone. Routine corrective gingivoplasty should not be needed post-operatively. It sounds as if the authors were commenting on a conventional flap operation.

I would also like to question the advisability of routinely incorporating terramycin into the surgical dressing and placing all patients on anti-biotics following mucogingivoplasty.

Claude Nabers
South Texas Building
San Antonio, Texas

References:

1. Ariaudo, A. A., and Tyrrell, H. S.: "Repositioning and Increasing the Zone of Attached Gingiva," Journal of Periodontology, Vol. 28, Number 2, pp. 106-110, 1957.

2. Nabers, Claude: "Repositioning the Attached Gingiva." The Journal of Periodontology. January, 1954, pp. 38-39.

UNIVERSITY OF MINNESOTA

The 2nd Annual Course in *ADVANCED ORAL PATHOLOGY* will be given by the University of Minnesota during the week of April 20-24, 1959. The Course consists of both lectures and laboratory. The subjects and faculty will include: Tumors of the Skin—Dr. *Herbert Z. Lund*, Professor of Dermatology, University of North Carolina; Tumors of Soft Tissues—Dr. *Raffaele Lattes*, Professor of Surgical Pathology, Columbia University; Skin Dermatoses—Dr. *Robert Goltz*, Assoc. Professor of Dermatology, University of Minnesota; Non-Neoplastic Diseases of the Jaws—Dr. *William Shafer*, Professor of Oral Pathology, Indiana University; Tumors of Bone—Dr. *David C. Dahlin*, Assoc. Professor of Pathology, Mayo Clinic; Radiation Pathology—Dr. *Donn Mosser*, Professor of Radiology, University of Minnesota; Advanced Roentgenographic Diagnosis—Dr. *Edward Stafne*, Professor of Dentistry, Mayo Clinic; Salivary Gland Pathology—Dr. *Anand Chaudhry*, Associate Professor of Oral Pathology, University of Minnesota; Odontogenic Lesions—Dr. *Robert Gorlin*, Professor of Oral Pathology, University of Minnesota.

The tuition will be $65.00. The Course registration is limited to 15 individuals. Housing for the five days will be approximately $15.00. Interested individuals should *immediately* contact: Dr. Robert J. Gorlin, Chairman, Division of Oral Pathology, School of Dentistry, 242 Owre Hall, University of Minnesota, Minneapolis 14, Minnesota.

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